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INFLAMMATION.

I. EXUDATIVE INFLAMMATION.

THE blood-vessels take the active share in the inflammatory process, and the elements of the blood, which escape from them, constitute the inflammatory products.

The steps of the process can be observed in the bladder and mesentery of the frog. The arteries and veins are dilated, the blood-current is slower; in the veins the peripheral layer of white blood-cells becomes thicker and slower; in the capillaries the white blood-cells accumulate. There is emigration of the white blood-cells; diapedesis of the red blood-cells, and exudation of serum rich in albumen, or in fibrino-plastic substances.

There must be changes in the walls of the blood-vessels, but we are ignorant of their nature.

VARIETIES OF EXUDATIVE INFLAMMATION. 1. *Simple acute exudative inflammation*.—The changes in the blood-vessels, and the production of serum, fibrin, and pus, are associated with few or no changes in the surrounding tissues.

a. *In connective tissue*.—Connective tissue is composed of a basement substance and of cells. Imbedded in it are the blood-vessels, nerves, and lymphatics.

There are congestion, swelling, and infiltration with serum, fibrin, and pus in variable quantities.

b. *In mucous membranes.*—The structure of these membranes is more complex than that of connective tissue. They are all composed of a layer of epithelium, of a connective-tissue stroma containing the blood-vessels, nerves, and lymphatics, and of mucus glands. There are congestion, swelling, emigration of white blood-cells, diapedesis of red blood-cells, transudation of serum. The quantity of inflammatory products is usually small. The production of mucus is at first arrested, afterward increased and altered. There may be excessive desquamation of epithelium and the formation of superficial ulcers. Such an inflammation is also called "Acute Catarrhal Inflammation."

c. *In the viscera.*—The viscera are all composed of a connective-tissue stroma containing the blood-vessels, lymphatics, and nerves, and of cells. The cells are peculiar to each viscus, and are concerned in performing the functions of the viscus. Congestion and swelling are present; the quantity of inflammatory products is usually moderate; there is derangement of the functions of the viscus.

The inflammation is of the same nature in connective tissue, the mucous membranes, and the viscera. The differences are due to the different structures of these parts. The process is always a transitory one, and does not change the tissues of the inflamed part. After the termination of the inflammation the congestion subsides, the inflammatory products are absorbed, and the parts return to their natural condition.

2. *Acute exudative inflammation, with excessive emigration of the white blood-cells.*—This is often called purulent inflammation. The excessive quantity of pus-cells may or may not be accompanied by serum and fibrin.

The inflammation is of a more severe type than is simple exudative inflammation.

Connective tissue is infiltrated with the inflammatory products.

In the mucous membranes the pus-cells are mixed with the mucus on the surface of the inflamed membrane.

In the viscera the stroma is infiltrated with the pus-cells.

3. *Exudative inflammation with degeneration, or death, of tissue.*—The changes in the blood-vessels, and the consequent congestion and production of serum, fibrin, and pus, exist, but there is added death or degeneration of parts of the tissues in which the inflammation exists. This additional change is apparently due to the presence of pathogenic bacteria.

In connective tissue there are congestion, exudation of serum, emigration of white blood-cells, and death of portions of tissue. The dead tissue softens, breaks down, and cavities are formed which contain serum, pus-cells, and portions of dead tissue. Such cavities, with their contents, are called abscesses.

In mucous membranes there are congestion, exudation of serum rich in fibrino-plastic substances, emigration of white blood-cells, and necrosis of tissue.

The fibrin infiltrates the stroma and coagulates on the surface of the mucous membranes, so as to form false

membranes. The pus-cells are entangled in the fibrin. The necrosis involves only the epithelium, which passes into the condition of coagulation necrosis and forms part of the false membranes ; or it involves also the stroma. The death of the epithelium forms superficial erosions, that of the stroma, ulcers of varying size and depth.

Such an inflammation of the mucous membranes is called "croupous," or "diphtheritic."

In the viscera the congestion is more or less marked, there are exudation of albuminous serum and emigration of white blood-cells. In addition there may be either swelling and degeneration of the visceral cells, or death of portions of the stroma with groups of cells, and the formation of abscesses.

This variety of exudative inflammation is of severe type, is accompanied with marked symptoms, and after it has subsided, leaves changes in the tissues.

In connective tissue the cavities of the abscesses are filled first with granulation tissue, and afterward with cicatricial tissue.

In the mucous membranes the dead epithelial cells may be replaced by new cells of the same kind, but the ulcers formed by the death of the stroma have to be filled first with granulation tissue, and later by cicatricial tissue.

In the viscera the degenerated cells may be replaced by new visceral cells, but the abscess cavities are filled first with granulation tissue, and later with cicatricial tissue.

4. *Exudative inflammation with the production of new tissue.*—The changes in the blood-vessels, and the forma-

tion of the inflammatory products, are well marked, but there is added from the first a growth of new tissue. This new tissue is at first of indifferent type, or like granulation tissue ; later it may take the form of connective tissue.

In connective tissue the fibrin, serum, and pus are found in varying quantities. The new tissue forms thickenings and adhesions.

In the mucous membranes the serum, fibrin, and pus are present only in small quantities. The function of the mucus-glands is at first arrested, and afterward increased ; the stroma is infiltrated with the new tissue.

In the viscera the quantity of the inflammatory products varies. The new tissue is produced in the stroma ; there may also be degeneration of the visceral cells. In the viscera this may be called a "diffuse" inflammation. The most marked features of this form of exudative inflammation are its tendency to persist for a long time, and the frequency with which it runs a subacute rather than an acute course.

II. PRODUCTIVE INFLAMMATION.

The blood-vessels take no active share in the inflammatory process. The changes begin and continue in the cells and basement substance of connective tissue, whether by itself or forming part of mucous membranes, or viscera.

In the connective tissue of the pia mater and peritoneum this inflammation may run an acute course, and the inflammatory product consists only of new cells resembling the normal connective-tissue cells of these

membranes. This may be called "cellular" inflammation.

Much more frequently, this form of inflammation in connective tissue runs a chronic course with the formation of indifferent tissue, or of dense connective tissue. In this way thickenings and adhesions are formed. It may happen that portions of this new tissue will degenerate, or be calcified. The slower examples of this form of inflammation are often called "sclerosis."

In mucous membranes the growth of new tissue is in the stroma. This is thickened, either diffusely, or in the form of polypoid growths. The layer of epithelium may be thickened or thinned. The mucus-glands are atrophied, or hypertrophied, or become cystic. The production of mucus is diminished, or increased, or altered. This condition in the mucous membranes is commonly called a "chronic catarrhal" inflammation.

In the viscera there is a growth of indifferent tissue, or of connective tissue, in the stroma. The visceral cells are compressed, or degenerated, or fatty, or disappear. The functions of the viscus are seriously interfered with. In the viscera this is often called an "interstitial" inflammation.

The most marked features of this form of inflammation are its slow course and its tendency to continue. The lesions of chronic productive inflammation, especially in old persons, are by some believed to be due to chronic degeneration.

Tubercular and syphilitic inflammations are varieties of productive inflammation. The special poisons of tuberculosis and of syphilis may indeed give rise to exu-

dative inflammations also. But tubercle tissue and gummy tumors are the most constant accompaniments of the tubercle bacillus and the syphilitic microbe.

Tubercle tissue is developed in connective tissue, in the mucous membranes, and in the viscera. It is composed of a basement substance, at first delicate, afterward firmer, which encloses large and small polygonal cells and giant cells. It possesses few or no blood-vessels, is of low vitality, and is apt to undergo cheesy degeneration. The tubercle tissue is arranged in little spherical masses, called tubercle granula, or in diffuse infiltrations. Its gross appearance is that of miliary tubercles, less frequently of larger tubercular plates, infiltrations, or tumors.

Gummy tumors are found in connective tissue, in mucous membranes, and in the viscera. They are composed of a basement substance, and of small round, oval, and fusiform cells. They possess few or no blood-vessels, and frequently undergo cheesy degeneration. Their gross appearance is that of irregular tumors of some size, but occasionally they are small and resemble miliary tubercles of small size.

With the formation of tubercle tissue and of gummy tumors, there is often associated an obliterating endarteritis of the smaller arteries of the tissues involved in the inflammation.

ACUTE DEGENERATION.

The introduction into the body of certain poisons, such as arsenic, phosphorus, and mercury, and of the poisons of the infectious diseases, is regularly followed

by acute degeneration or death of the cells of the viscera.

A small quantity of one of these poisons produces simply a swelling of the visceral cells.

A larger quantity not only causes the cells to become swollen, but they are also infiltrated with granules of albuminoid matter and of fat, and are prone to disintegrate and break down. To these changes may be added a secondary exudative inflammation.

In the more intense forms of poisoning there is a rapid death of the visceral cells. They either disintegrate and break down, or pass into the condition of coagulative necrosis. With such a rapid death of the cells a well-marked secondary exudative inflammation is often present.

THE DURA MATER.

THE dura mater is a dense connective-tissue membrane, which lines the inner surfaces of the cranial bones, forms an investing membrane for the brain, and encloses the large venous sinuses.

Its external surface is adherent to the bones and forms their periosteum. Its inner surface is lined with endothelium, and is in more or less close apposition with the pia mater.

All inflammations of the dura mater, all forms of pachymeningitis, are apt, therefore, to be associated with lesions of the bones, the pia mater and the venous sinuses.

ACUTE EXTERNAL PACHYMEINGITIS.

Causes.—The ordinary causes are inflammation of the external periosteum of the cranial bones, fractures, osteitis, chronic otitis, and suppuration in the orbit.

Lesions.—The inflammation is circumscribed and corresponds to the position of the exciting cause. There is a collection of pus and fibrine between the dura mater and the bone.

Symptoms.—By itself this lesion gives few symptoms except the localized pain. Its importance consists in the liability to the development of meningitis and thrombosis of the venous sinuses.

THE DURA MATER.

Treatment.—The proper treatment is to trephine the skull and treat the inflammation antiseptically, or to apply continuous cold.

ACUTE INTERNAL PACHYMEINGITIS.

Causes.—It is secondary to external pachymeningitis, to chronic otitis, to pyæmia, puerperal fever, the exanthemata, facial erysipelas, and chronic Bright's disease.

Lesions.—The inflammation is occasionally circumscribed, but much more frequently diffuse. The inner surface of the inflamed dura mater is lined with a layer of fibrine and pus. In most cases the inflammation extends to the pia mater. There may also be thrombosis of the venous sinuses.

The symptoms and treatment are the same as those of acute meningitis, or of thrombosis of the sinuses.

CHRONIC INTERNAL PACHYMEINGITIS.

Causes.—It may be secondary to diseases of the bones, but more frequently occurs without discoverable cause. It is especially frequent in persons whose health has been enfeebled by other causes. In New York it is often observed.

Lesions.—There is formed on the inner surface of the dura mater a layer of new tissue, usually over the convexity, either on one side or both. This new tissue is closely adherent to the dura mater, it is composed of connective-tissue cells, a scanty basement substance, and numerous blood-vessels. In many cases the lesion advances no farther than the production of this very thin and delicate membrane. In other cases, however, there

are hemorrhages into the new tissue, this becomes thicker and denser, and so a mass may be formed thick enough to compress the surface of the brain. This may be followed by a chronic inflammation of the pia mater. It occasionally happens that a single large hemorrhage from the vessels of the new tissue will cause death within a few hours or days.

Symptoms.—The lesser degrees of this form of pachymeningitis give no symptoms. A large hemorrhage may occur even in such mild cases and, with no premonition, at once give the symptoms of severe cerebral apoplexy, which continue until death.

In the advanced cases there is continuous dull headache, gradual impairment of intellect ; the walk becomes unsteady, the articulation confused ; there may be attacks of temporary loss of consciousness, drowsiness, and stupor.

The course of the disease is slow and the symptoms intermittent. It is said that some of the patients recover. Usually they continue to grow worse, but are apt to die of some intercurrent disease.

Treatment.—The only measures likely to be of service are those directed to the general health of the patient.

SYPHILITIC PACHYMEINGITIS.

Lesions.—The inflammation results in the production of gummy tumors, which are situated either in the outer or inner layers of the dura mater, or in both. At the same time there is more or less chronic meningitis. Not infrequently gummy tumors also exist in other parts of

the body, especially in the periosteum of the cranial bones and in the pia mater.

Symptoms.—Headache continuous, but with exacerbations, and often very severe. Attacks of unconsciousness, or of general convulsions. The intellect is impaired, there are alternating stupor and delirium, there is often vomiting.

The pressure of the gummy tumor on the brain produces, according to its situation, blindness, deafness, localized convulsions, paralyses or rigidity, hemiplegia.

Treatment.—If the disease has not advanced too far, the regular antisyphilitic treatment is of great service. Mercury and the iodide of potash, given alternately or together, may cause the entire disappearance of the symptoms.

THROMBOSIS OF THE VENOUS SINUSES.

Causes.—Primary. Marantic—diarrhoea, phthisis, cancer, prolonged suppuration. Cases without discoverable cause. Young infants and old persons are especially liable, but it may occur at any age. Secondary. From some inflammation situated near the sinuses—osteitis, fractures, pachymeningitis, meningitis, abscess of the brain, otitis; from the pressure of tumors; from erysipelas of the face.

Lesions.—The sinus is partly or completely filled. The thrombi are red or white and firm, or softened and puriform. The veins entering the sinuses are congested or contain thrombi. The corresponding portion of brain tissue is congested, oedematous, softened, and hemorrhagic. There is often a complicating meningitis. If

the thrombus is broken down, there may be infarctions in the lungs and in other parts of the body.

Symptoms.—There will be first the history of the preceding disease, or inflammation, then the symptoms of the thrombosis. These symptoms are due to the change in the brain tissue, the complicating meningitis, and the septicæmia. Headache may precede the other symptoms for weeks or months. When the disease is established there are headache, neuralgic pains in the face, restlessness or delirium alternating with stupor, general convulsions, vomiting, fever, prostration, the pyæmic condition. There may also be strabismus, paralysis or rigidity of the muscles of the neck, arm, or leg. If the thrombus is in the longitudinal sinus, there may also be congestion and œdema of the forehead and sides of the head, epistaxis, and in children prominence of the fontanelle. In the cavernous sinus, œdema of the eyelids and temples, swelling of the veins in the orbit, exophthalmos. In the lateral sinus, congestion and œdema over the mastoid process.

Histories.—Case I. A fairly healthy male, forty-eight years of age, for thirty-four days before death suffered from occasional headache. After a time there was added neuralgia of the left side of the face. After nine days there was severe right hemicrania. After three days more he was in bed with alternating stupor and delirium, the pulse 56, the temperature normal, no paralysis. Seven days before death the pulse was 99, the temperature 100° F. He became comatose, and so died.

There was a pinkish thrombus filling the straight sinus and the right lateral sinus. Over the posterior end of

the second right frontal convolution there was thrombosis of the large veins of the pia mater, the pia was infiltrated with pus and serum, part of this convolution was mottled with hemorrhages. There was no lesion in any other part of the body.

Case II. A woman, twenty-three years of age, for two months before her death was anæmic and troubled with headache. Five days before death the headache grew worse, there was vomiting, dry tongue, stupor, pulse 70, temperature 98° F. One day before death she was restless, there were twitchings of the muscles of the arms and legs, she was unable to swallow. Just before death the temperature rose from 100° to 107° F. There were red and white thrombi in the straight sinus, in both lateral sinuses, and in the veins of Galen. Both optic thalami were softened and hemorrhagic. No other lesions were found.

THE PIA MATER.

THE pia mater is the investing membrane of the brain. It is composed of lamellæ of connective tissue so arranged as to form a spongy membrane. Its free surface is covered with endothelium, and the lamellæ of which it is composed are also coated with connective-tissue cells. It contains numerous blood-vessels which are continuous with those of the cortex of the brain.

The pia mater may be the seat of acute, chronic, tubercular, or syphilitic inflammations. Its inflammations are called meningitis or leptomeningitis.

ACUTE MENINGITIS.

Lesions.—There are two anatomical forms, both of which give rise to the same symptoms.

1. Cellular meningitis. After death the pia is found congested, dry, lustreless. It contains little or no serum, fibrine, or pus. The inflammatory product is new cells of connective-tissue type formed on the surfaces of the lamellæ.

2. Exudative meningitis. The pia mater is infiltrated with serum, fibrine, and pus in variable quantities. There may be little collections of pus between the pia and brain. The cortex of the brain and the cranial nerves may be involved in the lateral ventricles. The lateral ventricles contain clear or purulent serum, and in chil-

dren the quantity of serum may be so great as to distend the ventricles. There is often added inflammation of the pia mater of the cord. Both forms of meningitis are regularly diffuse, involving the larger part of the pia, although the inflammation may be more intense either over the convexity or the base.

Causes. — Meningitis occurs without discoverable cause, after injuries and inflammations of the cranial bones, otitis, pachymeningitis. It complicates Bright's disease, pyæmia, pneumonia, the exanthemata, typhus and typhoid fever, rheumatism. It is the characteristic lesion of epidemic cerebro-spinal meningitis.

Symptoms. — The idiopathic cases. There may be a prodromic period characterized by headache, irritability, sleeplessness, vomiting, conjunctivitis, and general malaise, or the invasion may be sudden. Severe headache, frontal, occipital, or general, begins early in the disease and continues throughout. Restlessness or delirium and stupor alternate throughout the disease ; either one may be the more marked. General or unilateral convulsions may mark the invasion of the disease, or be developed at any time during its course.

General or local hyperæsthesia of the skin belong to the earlier days of the disease. Involuntary twitchings of the muscles of the face, and rigidity of the muscles along the vertebral column, are frequently present. At first there is photophobia, then insensibility to light, then blindness, often internal strabismus. The hearing is at first acute and painful, later dull.

Vomiting may mark the invasion of the disease, or be continued ; in some cases it is a marked symptom. The

tongue is at first coated and moist, afterward dry. Constipation is the rule, but toward the close of the disease there may be diarrhœa. The urine is diminished in quantity, and often contains small quantities of albumen.

The temperature ranges from 100° to 104° F., sometimes higher; it follows an irregular course. The pulse during the invasion is rapid, then becomes slow, and in fatal cases, toward the close of the disease is rapid and feeble. Toward the close of the disease, the patients may become completely unconscious.

In children the disease may follow the same course as in adults, or be characterized only by convulsions, stupor, and a febrile movement, or resemble tubercular meningitis. In young adults we sometimes see protracted cases resembling tubercular meningitis, but terminating in recovery.

The Duration of the disease is from thirty-six hours to four weeks; most of the cases last for one or two weeks.

The Prognosis is always serious, but yet a considerable number of patients recover.

The meningitis which follows injuries and inflammations of the cranial bones, otitis, or pachymeningitis, and which complicates Bright's disease and the infectious diseases, is accompanied with the same symptoms as have just been described; but these symptoms are preceded by those which belong to the original injury, inflammation, or disease.

Treatment.—The treatment consists in the general management of the patient, the use of means to control the inflammation, and in relieving the pain and discomfort. The patients are to be put to bed at once, even in

the prodromic period. They are to be kept from every exertion or excitement. The food should be entirely fluid, the constipation should be relieved by laxatives or enemata, the skin is to be kept clean.

Treatment directed to the inflammation is most efficacious during the early days of the disease. The most reliable measure is the application of continuous cold over the entire external surface of the cranium by the cold-water coil or ice-bags. Counter-irritation is less efficacious and more disagreeable.

Of drugs the best are calomel, sulphate of magnesia, iodide of potash, and opium. The calomel and sulphate of magnesia are given only for one or two days, in such doses as will act on the bowels. The iodide of potash and the opium are given throughout the active period of the disease, in moderate doses. To relieve the headache, restlessness, and delirium we employ cold to the head, the bromides in full doses, chloral hydrate, or opium.

CHRONIC MENINGITIS.

Lesions.—Either the pia mater over the base, or that over the convexity, or both, may be involved; the inflammation being more often localized than with acute meningitis. The pia mater is thickened and opaque, succulent, infiltrated with serum, fibrine, pus, and new cells. There may be adhesions between the pia mater and the dura mater. The cortex of the brain may show an increase of the neuroglia cells, softening, or sclerosis. The ventricles of the brain may contain an increased quantity of serum, and their ependyma may be thickened.

Causes.—Chronic meningitis belongs to adult life. It may be secondary to fractures and inflammations of the cranial bones, to pachymeningitis, to chronic Bright's disease, and to alcoholism. It may be associated with chronic endarteritis of the cerebral arteries. It occurs in persons who are badly nourished and of dissipated habits.

The Symptoms are the same as those which have already been described as belonging to chronic internal pachymeningitis. The disease is usually protracted over many years, its course interrupted by periods of apparent improvement. It terminates in insanity, or in death from exhaustion with cerebral symptoms.

The Treatment is the same as that for chronic pachymeningitis.

TUBERCULAR MENINGITIS.

I. *In Children. Lesions.*—The dura mater is usually unchanged, but sometimes there are miliary tubercles on its inner surface. The surface of the brain is flattened by the distention of the ventricles. In the pia mater we find miliary tubercles and inflammatory products, most frequently at the base of the brain, but sometimes over its entire surface. The tubercles are most numerous in the pia mater over and in the sulci.

The tubercles are very small and transparent, or larger and white. They are composed of simple aggregations of round and epithelioid cells, or of masses of tubercle tissue surrounding small arteries. Tubercle bacilli are present in moderate numbers. The adjoining small arteries are often the seat of obliterating endarteritis.

The accompanying inflammatory products are serum, fibrine, and pus, which infiltrate the pia mater in varying quantities; or small cells alone, which hardly change the gross appearance of the pia. The ventricles of the brain are much dilated and distended with serum. Their ependyma is thickened and contains very small tubercles. The brain tissue around the ventricles is often soft. There may be similar lesions in the pia mater of the spinal cord.

In young children the tubercular meningitis is regularly only part of an acute general tuberculosis, so that we find tubercles in many other parts of the body.

Causes.—The children affected are usually under five years of age. There is often a family history of tubercular disease. The acute general tuberculosis may follow a localized tuberculosis, or be primary.

It is to be remembered that in children, although we call the disease tubercular meningitis, it is really an acute general tuberculosis with meningeal symptoms.

Symptoms.—There may be a prodromic period, probably due to the development of the general tuberculosis, which usually lasts for only a few days, but may be protracted for several weeks. During this period there are loss of flesh and strength, slight evening fever, irritability, sleeplessness, frontal headache, vomiting, constipation, and a coated tongue. These symptoms are not continuous, but are interrupted by periods of improvement.

When the disease is fairly established the child is in bed, the face flushed, anxious, or apathetic. There are

headache, convulsions, stupor alternating with restlessness or delirium. During the periods of restlessness the child moans or cries out. The bowels are constipated, there may be vomiting. Photophobia, aphasia, and hyperæsthesia of the skin may be present. The pulse is slow in proportion to the temperature, and irregular; the breathing is often irregular; the temperature is raised and irregular, not always corresponding with the severity of the disease. The urine is scanty and contains albumen. At about the end of the first week, sometimes earlier or later, there are strabismus, inequality of the pupils, ptosis, changes in the optic disk. In the second week the stupor becomes more profound, the delirium more marked. There may be rigidity of the muscles of the back of the neck, the arms, and the legs; or local convulsions or paralyzes, which are transitory or permanent; automatic movements of the arms and legs; anæsthesia of the skin. The pulse becomes more frequent, and the temperature higher. There is no longer photophobia, but insensibility to light, with dilated pupils.

The children die in convulsions, or in coma, or become extremely feeble and emaciated; the urine suppressed, the breathing rapid, the temperature still high or below the normal.

It is said that, when the meningitis involves only the pia mater over the convexity of the brain, there is no strabismus and but little vomiting. Headache, delirium, convulsions, and rigidity of the muscles are the chief symptoms. During the course of the disease there are often short periods of improvement.

The Duration of the disease is from one to four weeks.

It is said to be shorter when the convexity alone is involved.

The Prognosis is very unfavorable.

Treatment.—When we remember that the disease to be treated is not merely a meningitis, but an acute general tuberculosis, it is evident that treatment can do nothing but alleviate the patient's suffering. There are, however, some cases, with active cerebral symptoms, in which the diagnosis is uncertain, and for these we may employ the same means that are useful in acute meningitis.

II. IN ADULTS. *Lesions.*—The changes in the pia mater are the same as in children, but the ventricles of the brain are not usually involved.

The meningitis is less often part of a primary general tuberculosis; it is more often a localized tubercular inflammation, or secondary to a localized tuberculosis already existing in some other part of the body.

Symptoms.—An adult may suffer from general tuberculosis, involving the pia mater, without symptoms of meningitis. He may have a primary, localized tubercular meningitis with marked symptoms. He may have a localized tuberculosis in some other part of the body, which has given few or no symptoms, and then suddenly develops the tubercular meningitis, as if it were a primary inflammation. He may have an acute general tuberculosis with marked cerebral symptoms. He may suffer from a well-marked localized tuberculosis, such as pulmonary phthisis, and develop a complicating meningitis with marked symptoms.

Most of the well-marked cases of tubercular meningitis

are in young adults, between the ages of fifteen and twenty-five years.

Symptoms.—The invasion is sudden, with chill, fever, headache, vomiting, and marked prostration; or gradual, with headache, malaise, but so little prostration that the patient does not go to bed. When the disease is established, the headache is severe and continuous. Alternating stupor and restlessness, sleeplessness, or delirium, conjunctivitis, photophobia, strabismus, ptosis, involuntary contractions of the muscles of the face, arms, and legs, hyperæsthesia of the skin, and vomiting, are regular symptoms. There is an irregular febrile movement. The pulse is sometimes slow, sometimes rapid; the urine contains albumen and casts.

Paralysis of the face, of the muscles of the throat, of the arm, or of one side of the body, may be developed later in the disease. The symptoms may be marked and continuous, the temperature high, and the patient die comatose in from seven to fourteen days; or the symptoms are less marked, the temperature is lower, the patients go on for weeks, and die in a typhoid condition.

The Prognosis is bad, and yet it must be admitted that some patients, who give a well-defined clinical history of tubercular meningitis, do eventually recover.

Treatment.—This is the same as that for the tubercular meningitis of children.

SYPHILITIC MENINGITIS.

Inflammations of the pia mater, due to syphilis, regularly behave in one of three ways:

1. They give the clinical history of an acute meningitis.
2. They give the history of a chronic meningitis.
3. They give the symptoms of an intra-cranial tumor.

The Treatment is the regular antisyphilitic course with mercury and the iodide of potash. In the cases which behave like an acute meningitis, the application of cold to the head may also be indicated.

THE BRAIN.

THE cortical motor tract of the brain is composed of the two ascending central convolutions, the paracentral lobule, and the anterior two-thirds of the superior parietal lobule. The upper third of this tract controls the motions of the leg, the middle third those of the arm, the lower third those of the face.

Destruction of any part of this motor tract causes paralysis of the opposite side of the body, with degeneration of the pyramidal tract, and rigidity of the limbs.

The number and position of the muscles paralyzed depend upon the extent and position of the lesion.

Irritating disease and stationary partial lesions of the same tract cause convulsive movements, beginning in the corresponding muscles. These movements may remain confined to these muscles, or may extend to all the muscles of the same side, or to the muscles of both sides of the body.

This local commencement of the convulsive movements is characteristic of cortical lesions.

Lesions of the third left frontal convolution impair or destroy the power of uttering language.

Lesions of the first left temporal convolution impair or destroy the power of understanding spoken or written language.

Lesions of the apical region of the occipital lobe, especially of the cuneus, produce hemianopia.

Extensive lesions of the cortex around the fissure of Sylvius, extending over much of the central, parietal, and temporal convolutions, may cause hemiplegia and hemianæsthesia, loss of all the special senses, with blindness of the opposite eye.

Lesions of the white matter of the hemispheres produce symptoms resembling those of lesions of the cortex or of the internal capsule, according to the position of the lesions near one or other of these portions of the brain.

The internal capsule. Lesions of the angle and the posterior segment are the most common causes of hemiplegia. There is paralysis of the lower part of the face, the tongue, the arm, the leg, and, if on the left side, temporary aphasia. There may also be hemianæsthesia and hemianopia.

The corpora striata. Lesions of the caudate or lenticular nucleus generally produce hemiplegia, which is not permanent unless the internal capsule is also damaged. Small or chronic lesions may not produce hemiplegia.

Lesions of the crura cerebri produce loss of motion and sensation of the face and limbs of the opposite side, and paralysis of the muscles supplied by the third nerve on the same side. There are seldom convulsions.

The pons Varolii. Unilateral lesions produce paralysis of the limbs on the opposite side, and of the muscles supplied by the fifth and sixth nerves on the opposite side. Bilateral lesions produce paralysis on both sides, difficulty in swallowing, and inarticulation. Convulsions are rare in chronic, but common in acute, lesions.

The medulla oblongata. Severe lesions cause sudden death. Lesser lesions cause paralysis of the muscles supplied by the hypoglossal, glosso-pharyngeal, and spinal accessory nerves (Gowers).

Hemiplegia of the face, tongue, arm, leg, and side of the thorax is due to lesions of :

The pons Varolii, the crura cerebri, the corpus striatum, with the internal capsule, the ascending central convolutions and the lobulus paracentralis.

Sudden paralysis of the arm and leg, without affection of the cranial nerves, is most often due to lesions of the paracentral lobule and upper part of the central convolutions ; less frequently to lesions in the medulla, pons, crura cerebri, and internal capsules.

Sudden paralysis of one arm alone usually indicates a lesion of the middle third of the ascending central convolution.

General convulsions, beginning always in the same group of muscles after paralysis, indicate a cortical lesion.

Aphasia. Impairment of the function of expressing thought by spoken or written language, indicates a lesion of the third left frontal convolution.

Impairment of the capacity of understanding spoken or written language, indicates a lesion of the first left temporal convolution (Nothnagel).

THE CEREBRAL ARTERIES.

The anterior cerebral arteries supply the first frontal convolution, the anterior two-thirds of the second frontal, the upper end of the anterior central, the orbital surface

of the frontal lobe, and the anterior part of the caudate nucleus of the corpus striatum.

The posterior cerebral arteries supply the occipital lobe, the lower part of the temporal lobe, the optic thalami, and the corpora quadrigemina.

The middle cerebral arteries supply the third frontal convolution, the posterior end of the second frontal, most of the anterior and posterior central, the orbital surface outside of the orbital sulcus, the parietal lobes, the island of Reil, the superior and middle parts of the temporal lobes, the corpora striata, the inner parts of the optic thalami, and the internal capsule.

The medulla and pons are supplied by branches from the anterior spinals, vertebrals, basilar, and posterior cerebrals.

The cerebellum is supplied by branches from the basilar and vertebrals.

INFLAMMATION OF THE CEREBRAL ARTERIES.—The arteries throughout the body are frequently the seat of chronic inflammation, which follows one of two anatomical types.

1. There is a growth of cells and basement substance from the inner surface of the wall of the artery, which obstructs the lumen of the vessel, or may occlude it entirely. This is called "obliterating endarteritis." It terminates by cutting off the supply of blood from the tissues belonging to the diseased vessel.

2. There is a growth of dense connective tissue in the inner coats of the artery, which renders the arterial walls thick and rigid, and projects in flat masses into the lumen of the vessel. The new tissue thus produced is prone to

degenerate ; it may be converted into granular matter, or infiltrated with the salts of lime. In this way the walls of the artery become thickened in some places and thinned in others, its lumen is irregular, its inner surface is roughened. This is called "deforming endarteritis." It results in the rupture of the diseased vessel, in the formation of aneurismal dilatations, in the closure of the arteries by thrombi, or in rendering the supply of arterial blood to the parts belonging to the diseased artery uncertain and irregular.

Either of these forms of endarteritis may involve at the same time many of the arteries in different parts of the body, or a single set of arteries in one part of the body.

Causes. Chronic endarteritis is especially common in persons over forty years old. It is frequently associated with chronic gout, constitutional syphilis, pulmonary emphysema, chronic Bright's disease, and chronic endocarditis. But it may also occur by itself and without discoverable cause.

The cerebral arteries are often the seat, either of obliterating endarteritis or of deforming endarteritis.

According to the development of the lesion, we have extravasations of blood from rupture, softening of portions of the brain from occlusion or thrombosis, the formation of aneurisms within the cranial cavity, or an irregular supply of blood to portions of the brain.

We see very frequently patients in whom no cerebral clots, or areas of softening, or aneurisms, or thrombi are to be found, but who yet exhibit marked, and even fatal, cerebral symptoms. The cause of these symptoms is ap-

parently the change in the walls of the cerebral arteries and the consequent irregularities of the circulation.

The symptoms presented by these patients are at first in the form of temporary attacks, which last for hours or days, and then disappear. Such attacks are repeated at irregular intervals during months or years, the patient presenting few or no cerebral symptoms during the intervals. As time goes on the attacks become more severe and more frequent. It is, however, possible for the patient to die during the first attack.

The temporary attacks are not all of the same character. There may be only headache, or anxiety, restlessness, and insomnia, or vertigo, or aphasia, or hemianopia, or loss of consciousness, or general convulsions, or spasmodic contractions of groups of muscles, or paralysis of one arm, of one leg, or of the whole of one side of the body, or loss of sensation in different areas of the skin.

The first attack may prove fatal, but the patient often continues to live for months or years. Even after recovering from the first attack, however, there is a perceptible change in the patient's mental and bodily condition, sometimes very marked. The attacks are repeated until finally the patient develops the symptoms of chronic meningitis, or becomes completely hemiplegic, or dies with general convulsions or coma.

Treatment. Apart from antisyphilitic treatment in the proper cases, the most important points to enforce are abstinence from all alcoholic drinks, and exercise in the open air.

CEREBRAL HEMORRHAGE.

I. HEMORRHAGE BETWEEN THE SKULL AND THE DURA MATER.—Extravasations of blood in this position are produced by severe blows or falls on the head.

Lesions. There is the external bruise or wound of the scalp, beneath this often a fracture of the bone, and beneath this a clot between the bone and the dura mater. There may also be laceration of the cortex of the opposite side of the brain, and after some days inflammation of the membranes near the clot.

The symptoms, therefore, are due to the shock of the injury, the pressure of the clot, the laceration of the brain, and the meningitis.

The shock often causes temporary unconsciousness.

The clot produces general convulsions, coma, or paralysis, according to its size and position.

The laceration of the brain may cause general convulsions.

The meningitis is attended with its regular acute symptoms.

In all the cases, after twelve to twenty-four hours, there is a rise of temperature.

Immediately after the receipt of the injury the patients are usually unconscious for some hours, although sometimes they are conscious and even able to walk for several hours. If unconscious, they may remain so only for a few hours, or until death. Later come the rise of temperature, general convulsions, hemiplegia, coma, the symptoms of meningitis.

Treatment. The only treatment likely to be of use is to make a sufficiently large opening in the skull and remove the clot.

II. HEMORRHAGE BETWEEN THE DURA MATER AND THE PIA MATER.—*Lesions.* The clot is found between the pia mater and the dura mater, over the convexity of one cerebral lobe, or over both lobes, or over the base of the brain. It varies in size from a thin layer to a mass large enough to compress the brain.

Causes. Such clots are found at all ages. They are due to blows or falls on the skull, to injuries received during parturition, to diseased arteries, to chronic internal pachymeningitis, to the rupture of aneurisms, and occur without known cause.

Symptoms. There may be a prodromic period, lasting for several days, characterized by heaviness of the head, vertigo, headache, mental slowness, and vomiting; or the attack may be sudden.

There is a good deal of variety as to the number and severity of the symptoms in the different patients.

Loss of consciousness is developed gradually or suddenly; it is complete, partial, transitory, or permanent.

The intelligence, if there is no loss of consciousness, may be unimpaired. Delirium is often present.

The speech remains intact, or is lost, or partly impaired.

There may be strabismus; the pupils may be dilated, or contracted, or unequal.

There may be general or local convulsions, either at the beginning of the attack, or later.

There may be no motor paralysis, or hemiplegia, or bilateral paralysis. The paralysis may be developed gradually or suddenly ; it may be incomplete or complete, transitory or permanent. It is not as constant as with cerebral hemorrhage.

Sensation may be unchanged or impaired.

The pulse may be slow or rapid. There is regularly a febrile movement after the first twenty-four hours.

Vomiting often accompanies the beginning of the attack.

The following are some of the attacks which I have seen :

Large clots at the base producing sudden death.

Large clots over the whole of one hemisphere, with delirium, coma, a rise of temperature, and death in twenty-four hours.

A clot over the convexity of both frontal lobes, with bilateral paralysis, but without loss of consciousness.

A clot over the convexity of the right hemisphere, with convulsions, delirium, dilated pupils, coma, high temperature, death after several days. The symptoms resembled those of an acute meningitis.

A clot over the whole of the right hemisphere, with, for twenty-four hours, coma, contracted pupils, pulse 140, urine albuminous, and partial paralysis of the left arm and leg ; then for five days improvement, and then sudden death.

A clot over the whole convexity of the brain, with repeated convulsions, and death in twenty-four hours.

A clot over the whole convexity, with alternating coma and convulsions, and albuminous urine ; the whole attack resembling one of uræmia.

The Prognosis seems to depend upon the size of the clot.

The Treatment is symptomatic.

III. HEMORRHAGE BETWEEN THE PIA MATER AND THE BRAIN.—This is usually only an accompaniment of cerebral or meningeal hemorrhage, but it may occur by itself. When this is the case the clot is frequently situated at the base of the brain, and causes death within a short time.

IV. HEMORRHAGE IN THE SUBSTANCE OF THE BRAIN.—*Lesions.* The clots are small or large, usually single, sometimes multiple, not infrequently repeated at intervals. The blood forms a cavity for itself by lacerating the brain tissue ; it may break through into the cavity of the ventricles, or beneath the pia mater. With a large clot the surface of the brain is somewhat flattened and the pia mater dry.

If the hemorrhage is due to traumatism, we find the evidences of this ; if not, we find diseased arteries or miliary aneurisms.

If the patient continues to live, the clot shrinks and there is more or less inflammation of the surrounding brain tissue. Finally, there remains only a connective-tissue cicatrix, or a cyst.

Causes. Cerebral hemorrhage may be caused by injuries inflicted on the skull ; more frequently it is due to the rupture of diseased arteries, or of miliary aneurisms. The tendency to rupture seems to be increased by high arterial tension. With cerebral apoplexy we often find associated atrophied kidneys, and hypertrophy of the left ventricle of the heart.

The greatest liability to cerebral hemorrhage is between the ages of seventy-one to eighty, next in order from sixty-one to seventy, fifty-one to sixty. The least liability is between the ages of one to ten years.

Symptoms. Excluding the traumatic cases, a prodromic period is not uncommon. It is characterized by emotional irritability, dulness of the mental faculties, temporary attacks of drowsiness or loss of consciousness, temporary paralysis of the arm or leg, disturbances of sensation, and hemorrhages in the retina. All these symptoms are apparently due to the chronic inflammation of the cerebral arteries which already exists.

The symptoms at the time of, and just after, the hemorrhage depend upon the size and position of the clot, and the rapidity with which the blood escapes from the ruptured vessels.

Large clots in the corpora striata, internal capsule, crura cerebri, and central convolutions produce sudden coma, hemiplegia with facial paralysis, and death within a few days.

Clots in the pons Varolii, medulla oblongata, and corpora quadrigemina produce sudden coma, hemiplegia, and death.

Large clots outside of the motor tract and the basal ganglia produce alternating convulsions and delirium, coma, and death within a few days.

Smaller clots in the corpora striata and internal capsule, if they are formed rapidly, cause temporary unconsciousness, hemiplegia, and when on the left side, aphasia.

If they are formed gradually the mind is confused, the speech is affected, there is vomiting, and a gradual development of coma, hemiplegia, and aphasia.

Small clots in the central convolutions give no, or temporary, loss of consciousness, with localized paralyses of the face, arm, or leg.

Small clots in the third left frontal convolution give no, or temporary, loss of consciousness, with aphasia, either partial or complete.

Small clots outside of the motor tract may give temporary loss of consciousness, without paralysis.

In all the cases the pupils may remain natural, or be dilated, or contracted, or unequal. There is regularly deviation of both eyes toward the non-paralyzed side.

The pulse is usually slow and full. The temperature is at first lowered, but after twenty-four hours rises above the normal.

After two or three days, with the secondary fever, there are headache, stupor, delirium, congestion and œdema of the lungs, and rigidity of the paralyzed muscles.

After the lapse of weeks or months some of the patients recover entirely, some show failure of the mental faculties and emotional irritability, in some the motor paralysis persists either completely or incompletely. The paralyzed muscles are often contracted, the limbs swollen and painful.

Prognosis. Complete coma lasting more than twenty-four hours, pronounced congestion and œdema of the lungs, bilateral paralysis, a very low initial temperature, a high secondary temperature with delirium and stupor, all indicate an early and fatal termination of the disease.

For months afterward there is danger to life from meningitis, pneumonia, cystitis with suppurative nephritis, and bed-sores.

If the patients do not die and are to do well, there should be some improvement in the paralysis, the aphasia, and the mental condition within a month after the commencement of the attack.

Some patients, even with small clots, do badly on account of the changes in the cerebral arteries.

The Treatment is symptomatic.

EMBOLISM AND THROMBOSIS OF THE CEREBRAL ARTERIES.—*Lesions.* Apart from septicæmia, most of the cerebral emboli come from the aortic and mitral valves, the endocardium of the left ventricle, and the arch of the aorta.

Most frequently there is a single embolus in one of the branches of the left, or right, cerebral artery. Less frequently the plug is in the anterior or posterior cerebrals, or emboli find their way into different arteries at different times, or one of the large arteries at the base of the brain is occluded, or all the arteries are filled at once with a mass of detritus.

Thrombi are found most frequently in the internal carotids, middle cerebrals, basilar, vertebral, and posterior cerebral arteries.

Occlusion of an artery, either by an embolus or a thrombus, is followed by anæmia, or less often by venous congestion of the corresponding portion of brain tissue. After about twenty-four hours this portion of brain degenerates and softens; finally it is replaced by a cicatrix or a cyst with fibrous walls.

The softened areas of brain are found in the cortex, corpora striata, optic thalami, pons Varolii, medulla oblongata, and cerebellum.

Causes. The regular causes of cerebral embolism are endocarditis affecting the aortic or mitral valves, or the endocardium of the left ventricle ; or chronic inflammation of the arch of the aorta. But it must be admitted that in some cases we cannot find the source of the embolus.

Cerebral embolism occurs in both sexes. It is most frequent between later childhood and middle age, but is not uncommon in older persons.

Thrombosis is due to chronic endarteritis, or to changes in the blood and the circulation. The puerperal condition, the infectious diseases, and the chronic exhausting diseases may be complicated by thrombosis.

Symptoms. The symptoms of embolism are developed suddenly.

Those of thrombosis may be preceded by the symptoms of cerebral endarteritis, and may be developed suddenly or gradually.

The sudden plugging of most of the cerebral arteries causes sudden death.

An embolus filling one of the large arteries causes coma, hemiplegia, and death within a few days.

Emboli in one of the smaller arteries produce symptoms at the time when they lodge, by their sudden interference with the functions of a portion of the brain tissue ; later, by the death of the same portions of the brain.

At the commencement of the attack the symptoms vary with the size and position of the artery occluded.

With embolism loss of consciousness is less frequent and of shorter duration than is the rule with cerebral hemorrhage.

With thrombosis loss of consciousness is said to be less frequent than with embolism.

General convulsions occur oftener with embolism than with thrombosis. Vomiting often accompanies the attack. There is usually no change in the temperature, or only a slight fall. In the patients, who do not lose consciousness, there may be headache, vertigo, incoherent speech, delusions, or hallucinations.

According to the position of the occluded artery, we have motor paralysis, convulsive movements, or rigidity affecting only the arm, face, or leg, or one side of the body, or both sides.

If the third left frontal convolution is involved, there is motor aphasia; if the first left temporal convolution, there is sensory aphasia; if the posterior part of the occipital lobe, there is hemianopia.

After from twelve hours to four days there is a rise of temperature, sometimes accompanied with other symptoms, due to the development of inflammation around the area of softening.

After two or three days there is often a temporary improvement in the paralysis or aphasia.

Later in the disease the patients may :

1. Continue comatose, hemiplegic, and die.
2. Remain permanently hemiplegic, with or without aphasia. The mental faculties return nearly to the normal, or are impaired. The paralyzed muscles often become contracted, there may be general convul-

sions, the patients may develop a complicating meningitis.

3. Recover, either partly or completely, the power of speech and of voluntary motion. It is to be noted that in some of these apparently mild cases there is marked and permanent impairment of the mental faculties.

4. Recover either partly or completely, but have subsequent attacks of the same character.

Diagnosis. Cerebral embolism differs from cerebral hemorrhage in that :

It occurs regularly in younger people. The lesion is apt to be smaller, situated in the cortex and on the left side.

Loss of consciousness is less constant and more temporary ; local paralyses and aphasia alone are more common.

Prognosis. The lesion seems to be less fatal than is cerebral hemorrhage, but yet complete recovery is not so very common.

The Treatment is symptomatic.

ABSCESS OF THE BRAIN.

The small multiple abscesses of the brain, which occur with pyæmia, form part of that disease and require no separate description.

The large single abscesses, occurring under different conditions, are those to which the names of " Abscess of the Brain " is usually applied.

Lesions. These abscesses occur in two forms :

The non-capsulated abscess, an irregular cavity con-

taining a thin pus and softened brain tissue. The walls of the cavity are ragged and infiltrated with pus, and outside of the walls is a zone of œdematous and softened brain tissue. If the abscess is near the pia mater it may set up a meningitis ; if it is near the lateral ventricles it may rupture into them ; if it is near the sinuses of the dura mater it may cause thrombosis.

The encapsulated abscess has a capsule of connective tissue, and contains thin or cheesy pus.

Abscesses of the brain are usually single ; they may attain a considerable size. They are most frequent in the cerebral and cerebellar hemispheres, rare in the central ganglia, the pons, and the medulla oblongata.

Causes. The most common cause of this disease seems to be chronic suppurative otitis (42.5 per cent., Gowers), while acute otitis is a comparatively rare cause. With the otitis there may also be caries of the temporal bone, suppuration of the mastoid cells, and inflammation of the dura mater. The abscess is usually situated deep in the brain, only rarely is it continuous with the inflamed dura mater and bone. When the abscess is deeply situated, and the bone and dura mater are not diseased, it is difficult to tell how the infection travels from the ear to the brain. Abscesses due to this cause are situated in the temporo-sphenoidal, the frontal, the occipital, and the parietal lobes, or in the cerebellum.

Another frequent cause of abscess of the brain is traumatism—blows or falls on the head (twenty-four per cent., Gowers). Such injuries may not hurt the skull, or may produce fractures, or necrosis. There is often a considerable interval between the time when the injury

is inflicted and that when the symptoms of the abscess are developed.

When the bones are uninjured, the abscess is situated deep in the brain; when there is necrosis of the bones, the abscess may be superficial; when the bone is fractured, the abscess may be either superficial or deep. The abscess is regularly situated beneath the point of injury, rarely in the opposite side of the brain.

Chronic disease of the nose, either the mucous membranes or the bones, has been the cause of a few abscesses in the frontal lobes. Disease of the orbit has also given rise to abscesses in the same position. In a few cases the abscesses have been due to caries of various portions of the cranial bones.

In a considerable number of cases ($\frac{1}{6}$, Gowers) no cause for the abscess has been discovered.

Symptoms. There are general symptoms common to all abscesses of the brain, and local symptoms due to the precise position of each abscess.

With the acute, non-capsulated abscesses the inflammation and the symptoms go on acutely until the death of the patient, the disease usually running its course within four weeks.

With the chronic, encapsulated abscesses, the course of the disease is much longer, and may be conveniently divided into an initial, a latent, and a terminal period.

The initial symptoms may be absent altogether; they may be mild, or they may be severe. They are especially marked with the traumatic cases. When well developed they resemble an acute meningitis; headache, local or general, general convulsions, a febrile move-

ment, stupor and delirium, vomiting, and prostration.

The latent period may last for months or years. There may be no symptoms during its course, or headache, mental disturbances, and general convulsions, which come and go.

The terminal stage is developed gradually, or suddenly. Rarely, the patient dies from some intercurrent disease before he has reached the terminal stage.

Usually, however, the symptoms of the terminal period are well marked. There are headache, vomiting, optic neuritis, general convulsions, alternating stupor and delirium, a slow pulse, a febrile movement often with chills. The patient passes into the typhoid state.

The local symptoms. Abscesses situated in the frontal lobes give no local symptoms.

Those situated in the motor tract cause paralysis, convulsive movements, or rigidity of the corresponding muscles.

Those situated in or beneath the speech tract cause motor or sensory aphasia.

Those situated in the occipital lobes may produce hemianopia.

Those situated in the cerebellum are very often latent.

The Diagnosis is between such abscesses, tumors of the brain, and acute meningitis.

The Prognosis is unfavorable.

Treatment. It is to be remembered that chronic otitis is the most common cause of abscess of the brain, and that for this reason special attention should be given to the treatment of such an otitis.

While the symptoms are acute the treatment is the same as for acute meningitis.

When the abscess is so situated that it can be reached, surgical interference has proved very satisfactory in a number of cases.

TUMORS OF THE BRAIN.

INTRACRANIAL tumors are syphilitic, tubercular, gliomata, sarcomata, fibromata, osteomata, or chondromata. There are also found in the brain cysticercus cysts and hydatid cysts.

The syphilitic gummy tumors are single or multiple. They originate in the dura mater or pia mater, and compress or invade the brain. They are, therefore, regularly cortical tumors, most frequently situated over the cerebral hemispheres.

The tubercular tumors are often multiple. They may constitute the only tubercular lesion in the body, or there may be similar lesions elsewhere. They are regularly formed in the brain-tissue, and are found in the cerebellum, cerebrum, pons, central ganglia, crura cerebri, medulla oblongata, and corpora quadrigemina.

The gliomata are composed of tissue resembling the neuroglia, but with an excess of cells. They are often soft and hemorrhagic. They are usually single, originate in the brain-tissue, and may be found in any part of the brain.

The sarcomata are composed of connective-tissue, with an excess of round, oval, and fusiform cells. They

originate in the brain-tissue, the membranes, and the cranial bones, and compress or infiltrate the brain. They are usually single, but may be multiple.

Sarcomata, composed of flat endothelial cells arranged in alveoli, originate in the pia mater. They are usually single, but may be multiple, and in rare cases are found in large numbers on the dura mater and pia mater, both of the brain and spinal cord.

Causes.—Syphilitic tumors may be developed at any time from twelve months to fifteen years after the initial lesion. They are said to be most frequent at intervals of from five to twelve years.

Tubercular tumors are most common in persons under twenty years of age. There may be a family history of tuberculosis, or tubercular lesions in other parts of the body.

The gliomata and sarcomata are most common in persons between the ages of twenty-four and forty years.

The cysticercus and hydatid cysts are found regularly between the ages of ten and twenty years.

Symptoms.—It is possible for a tumor of the brain to give no symptoms at all. The patient dies from some other disease and the tumor is discovered at the autopsy.

In a few cases, although the tumor has existed for some time, no symptoms are developed until just before the patient's death.

Ordinarily the presence of the tumor is attended with symptoms. There are general symptoms common to all intracranial tumors, and local symptoms due to the position of each special tumor.

1. The general symptoms. Headache is rarely ab-

sent. It is usually severe, either localized or general, often constant, with exacerbations, sometimes intermittent.

Optic neuritis is a frequent symptom. The retinal veins are swollen, the optic disc and retina are infiltrated and swollen, there are extravasations of blood in the retina. Later these changes are followed by atrophy of the optic disc. Such an optic neuritis may be developed with any intracranial tumor. It is less frequent with tumors of the membranes only compressing the convexity of the hemispheres, than with tumors involving the substance of the brain. The degree of disturbance of vision varies in the different cases. It is most marked with hemorrhage and after the nerve has become atrophied.

Stupor and coma belong to the later stages of the disease.

Gradual failure of the mental powers, loss of memory, depression, emotional excitement, delusions, or hysterical phenomena are often present.

General convulsions followed by temporary unconsciousness occur in some cases.

Vertigo, either constant or paroxysmal, may exist with tumors in any part of the brain. It is most marked with tumors of the pons, the corpora quadrigemina, and the cerebellum.

Vomiting is especially common with tumors of the medulla, the middle lobe of the cerebellum, and the corpora quadrigemina. With cerebellar tumors vomiting, headache, and optic neuritis are often, for a time, the only symptoms.

Slowness of speech and difficulty in articulation may exist with tumors of the pons, the medulla, or the posterior fossa at the base of the brain.

Temporary loss of consciousness is a symptom of some intracranial tumors.

The local symptoms.

Motor aphasia is produced by tumors of the third left frontal convolution, sensory aphasia by tumors of the first left temporal convolution.

Paralysis.

1. Gradual development of hemiplegia belongs to tumors in, or compressing, the pons, the crura cerebri, and the internal capsule.

2. Paralysis of the arm, the arm and face, or the leg is associated with tumors in or beneath the cortical motor tract. The paralysis begins in the hand or foot.

3. Bilateral paralysis is produced by symmetrical tumors on both sides of the brain, or by a single tumor compressing both motor tracts. The paralysis, however, is not usually equally developed on both sides of the body.

The paralyzed muscles are often contracted.

There may be convulsive movements of the muscles of the face, hand, or foot, and extending thence to the adjoining muscles.

There may be feelings of pain, tingling, and formication in the paralyzed or convulsed limbs.

Hemianæsthesia may be produced by tumors involving the posterior part of the internal capsule.

The cranial nerves are compressed and their functions interfered with by tumors situated at the base of the brain.

The duration of life with intracranial tumors is considerable, many of the patients live for years.

The treatment varies with the character and position of the tumor. Syphilitic tumors require the regular treatment with mercury and the iodide of potash. Tubercular tumors require the same management as chronic phthisis. Tumors of any nature in accessible situations are proper subjects for surgical operation and removal.

INTRACRANIAL ANEURISMS.

Lesions.—These aneurisms are usually sacculated, from the size of a pea to that of a pigeon's egg. The relative frequency of their origin is given by Gintrac as follows : Basilar artery, 20 ; middle cerebral arteries, 18 ; internal carotid arteries, 17 ; anterior cerebral arteries, 7 ; vertebral arteries, 5 ; posterior communicating arteries, 5 ; cerebellar arteries, 4 ; posterior cerebral arteries, 3 ; anterior communicating arteries, 2 ; arteries of corpus callosum, 2.

Rather more than half of these aneurisms rupture within the cranial cavity.

Symptoms.—The smaller aneurisms may give no symptoms ; or the first symptoms will be those of meningeal apoplexy from their rupture. The larger aneurisms give the symptoms of tumors at the base of the brain.

Causes.—These aneurisms, like others, are due to chronic endarteritis. They are most frequent in males between the ages of thirty-four and fifty-one years.

Treatment.—The only plans of treatment, so far known, seem to be the systematic administration of the iodide of potash, or ligature of the carotid artery.

THE VENTRICLES OF THE BRAIN.

Inflammations of the pia mater are often accompanied by lesions of the ventricles of the brain. Less frequently we find lesions of the ventricles occurring by themselves, with little or no change in the pia mater. We distinguish :

Acute endymitis.

Acute dilatation of the ventricles in adults.

The chronic hydrocephalus of children.

Chronic hydrocephalus due to injuries, or secondary to meningitis.

1. *Acute Ependymitis*.—Of this we find two varieties : an acute and a subacute form.

In the acute form we find the ependyma congested, infiltrated with pus-cells, and coated with a layer of fibrine and pus. The ventricles contain purulent serum.

The patients exhibit a decided febrile movement, general convulsions, alternating stupor and delirium, local paralyses, and finally coma. The disease runs its course within a few days. The symptoms resemble those of an acute tubercular meningitis.

So few of these cases have been observed that we are ignorant of the prognosis.

The treatment would seem to be the same as that for acute meningitis.

In the subacute form the ventricles are dilated and full of serum. The ependyma is thickened, the endothelial cells on its surface are multiplied, and there is a growth of cells around the blood-vessels.

The patients have headache ; vomiting ; at first pho-

tophobia, later dilated pupils ; hyperæsthesia of the skin ; an irregular rise of temperature, never very high, and sometimes below the normal ; finally coma. The disease lasts for weeks or months. The symptoms resemble those of a subacute tubercular meningitis.

These cases again are so seldom recognized that we do not know how many of them may recover.

2. *Acute Dilatation of the Ventricles in Adults—Serious Apoplexy—Lesions.*—Either one lateral ventricle, or both, or all the ventricles are dilated and filled with clear serum. The ependyma is white, somewhat thickened, its surface smooth or finely granular. The convolutions of the hemispheres are somewhat flattened.

Causes.—The disease is, in New York, not an uncommon one. It occurs both in strong and vigorous adults and in persons who have suffered from chronic diseases. In some persons there is a previous history of injuries to the head, or of mental or bodily overwork. In some cases the condition is complicated by chronic endarteritis, or chronic meningitis. It is said that there may be an obstruction of the veins or of the passages between the ventricles.

The disease may be confounded with cerebral apoplexy, embolism, chronic endarteritis, or tumors of the brain.

Symptoms.—The patients at first suffer from attacks of headache, noises in the ears, loss of memory, dulness of the mental faculties, slowness of speech, sleeplessness, disturbed digestion, sometimes temporary aphasia, or local paralyses. These symptoms come and go for from one to four weeks, rarely they only last for twelve hours.

Then there is a sudden change in the condition of the patients.

Some of them either gradually or rapidly pass into the condition of complete coma. In this condition they remain and die in a few days, the temperature rising before death.

Others develop an acute delirium, blindness, internal strabismus, convulsive movements of some of the muscles, a febrile movement, and finally become comatose.

3. *The Chronic Hydrocephalus of Children—Lesions*—There is an accumulation of serum in the ventricles of the brain, so large in quantity that the brain is thinned, the dura mater stretched, and the cranial bones separated from each other. The ependyma appears to be normal, or is somewhat thickened.

Causes.—We are ignorant of the causes of this lesion. It seems to begin during intra-uterine life. It may reach a considerable development before birth, or remain latent for months or years after birth.

Symptoms.—If the disease is so much developed during intra-uterine life that the large head prevents the birth of the child, craniotomy must necessarily be performed.

If the disease is moderately developed during intra-uterine life, the child can be born alive. But it is at once evident that the head is too large; the child does not perform its functions well, and dies in a short time.

In many cases at the time of birth the child shows no evidences of the disease, and it is not till after months or years that its symptoms are developed. Of these later developed cases we may distinguish a mild and a severe form of the disease.

In the mild cases the head is large, the fontanelles and sutures open, the face small. The intelligence remains good. From time to time, for periods of several days, the child suffers from disturbances of digestion and nutrition, a febrile movement, fits of crying, restlessness, and drowsiness.

In these mild cases the disease is not of itself fatal, but the children are apt to be carried off by some of the diseases of childhood.

In the severe cases there is marked impairment of nutrition; the children are dull and stupid, irritable and drowsy; there are often strabismus, nystagmus, dilated pupils, protruded eyeballs, blindness, deafness, general convulsions, and paraplegia. There may be a febrile movement. These symptoms are interrupted by periods of improvement, but the course of the disease is usually downward and the children die in an attack of convulsions, or of inanition, or from some intercurrent disease. Rarely they grow up to adult life, but remain more or less idiotic.

The treatment is mainly directed to the general condition of the patient. Careful feeding, a clean skin, good air, the administration of iron and of cod-liver oil are the essentials. During the exacerbations of the symptoms the iodide of potash may be of service.

4. *Chronic Hydrocephalus in Older Children.*—In older children and in adolescents we meet with cases of chronic hydrocephalus, not congenital, but occurring after meningitis, after injuries, and without discoverable cause.

In some cases of acute meningitis the patient, after going through the symptoms of this disease and begin-

ning to improve, makes only an attempt at convalescence, again becomes worse, is alternately delirious and stupid, has a moderate febrile movement and emaciates. After continuing in this condition for weeks, he may recover or die.

In the cases which occur after injury and without discoverable cause the clinical history is like that of the chronic hydrocephalus of young children, but without the enlargement of the head.

THE SPINAL CORD.

The spinal cord is composed of the gray matter, consisting of nerve-cells and fibres, with a very delicate connective-tissue stroma; and the white matter, composed of nerve-fibres, held together by a more abundant stroma.

The gray matter is divided into the anterior and posterior cornua, from which are given off the anterior and posterior nerve-roots.

The white matter is divided into a number of columns.

The cord acts:

1. As a conductor from the brain to the peripheral nerves. Motor impulses originate in the brain and travel downward through the cord to the motor nerves. Sensory impulses originate in the sensory nerves, and travel upward through the cord to the brain.

Disease of the motor tract—the pyramidal columns, the anterior gray cornua, and the anterior nerve-roots—causes loss of motor power.

Disease of the sensory tract—the antero-lateral columns and the posterior median columns—causes loss of sensation to pain and to touch.

Irritation of the posterior nerve-roots gives rise to pain referred to the peripheral sensory nerves.

2. As a series of centres for reflex action.

Loss of reflex action is due to interruption by disease of some part of the reflex arc at any point between the peripheral endings of the sensory and motor nerves.

Excess of reflex action is due to irritation of the nerve-centres, or to disease between the centres concerned and the brain.

3. It contains centres that ultimately govern nervous action in structures under the immediate control of the sympathetic system, such as the bladder and the rectum.

4. It controls nutrition.

Disease of the motor nerve-cells, or fibres, is followed by wasting of the muscles.

MYELITIS.

Lesions.—The stroma of the cord is changed into gelatinous matter, the nerve-cells and fibres degenerate, there is an exudation of serum, an emigration of white blood-cells, and a diapedesis of red blood-cells. All these changes, however, are not present in every case of myelitis. Either the exudation or the degeneration of the nerves, or the changes in the stroma, may constitute the principal or the only part of the lesion. In some cases the walls of the arteries are thickened.

The affected portions of the cord are either swollen, or softened and diffuent.

The lesions may involve the whole length of the cord—diffuse myelitis; or only a segment of the cord—transverse myelitis.

Causes.—Injuries, exposure, over-fatigue, the infectious diseases, and sexual excesses are often assigned as causes, but many cases occur for which no cause can be discovered.

Symptoms.—1. There are cases of myelitis characterized by an acute development of paraplegia, with constitutional symptoms.

The patient is suddenly attacked with vomiting, headache, delirium, a febrile movement, marked prostration, sometimes blindness. At the same time loss of motion and sensation, and increased reflex action are rapidly developed in the portions of the body situated below the lesion of the cord. The disease runs a rapid course and terminates fatally.

Such a severe and rapid course of the symptoms belongs to the forms of myelitis with the production of pus, and to those which are diffuse.

2. There are cases characterized by a rapid development of paraplegia without constitutional symptoms.

The patient, while in his ordinary health, finds that he is losing power in his legs, and within twenty-four hours is completely paraplegic. Such a myelitis is not usually fatal, unless it is followed by an ascending myelitis extending up the cord.

3. There are cases characterized by a gradual development of the local symptoms without any constitutional disturbance.

First, there are disturbances of sensation in the parts which are afterward to be paralyzed; numbness, tingling, formication, pricking, etc. Then there are gradual loss of muscular power and of sensation, a feeling of con-

striction around the body, paralysis of the bladder and rectum, and increased reflexes.

These symptoms may occupy weeks or months before they terminate in complete paraplegia. They may advance continuously, or with intermissions.

The myelitis does not of itself usually cause death, but life may be destroyed by bed-sores, by cystitis with suppurative nephritis, or by pneumonia.

Treatment.—While the disease is active the patients should be kept in bed, with the most careful attention to the skin and the bladder. If constitutional symptoms are present, the patients should be kept on fluid diet, otherwise they may eat ordinary food.

If the myelitis is of acute type, we may apply wet or dry cups, continuous cold or hot douches over the spine, and give internally calomel, sulphate of magnesia, or opium.

If the myelitis is of subacute type, we may use hot douches over the spine, and give internally iodide of potash, ergot, belladonna, nitrate of silver, the chloride of gold and sodium, arsenic, or strychnia.

When the disease has run its course the patient should be out of bed and out of doors as much as possible. No further treatment of the disease is necessary, but much attention should be given to the bladder, and to the skin and muscles of the paralyzed parts. Massage, passive motion, and electricity are now of real service.

ANTERIOR POLIO-MYELITIS.—INFANTILE PARALYSIS.—*Lesions.*—There is an inflammation of exudative type, with congestion and emigration of the white blood-cells. The nerve-cells and fibres may remain unchanged, or a small number of them may degenerate and die. When

the disease has run its course the cord may return to its normal condition, or a small part of it may be permanently destroyed. The inflammation involves only a part of the anterior cornua, and is usually limited to a segment of the cord, but is sometimes diffuse.

Causes.—The disease occurs most frequently in young children during the period of dentition. In some cases there is a history of exposure, or of injury, or of excessive fatigue. The attacks are more frequent in summer than in winter. Healthy children are as liable to the disease as are others. It is said that it may complicate the exanthemata, typhus and typhoid fever, bronchitis, and pneumonia.

Symptoms.—The invasion of the characteristic symptom—the paralysis—is acute or subacute. The paralysis may, or may not, be accompanied by constitutional symptoms.

The constitutional symptoms are : Fever, with the accompanying headache, loss of appetite, nausea, vomiting, restlessness, and prostration ; cerebral symptoms—general convulsions, alternating delirium and stupor ; disturbances of sensation—tingling, formication, pain along the nerves, in the muscles, and in the joints.

The motor paralysis involves some or all of the muscles of one or more of the limbs, less frequently the muscles of the neck and throat. The muscles of the bladder and rectum are but seldom involved. The full development of the paralysis may be effected within a few hours, or may require days or weeks. After reaching its full development the paralysis remains stationary for from two to six weeks, and is then followed by more

or less return of motor power. The return of motion begins in the muscles which were paralyzed the latest. The improvement continues for weeks or months, and is finally complete or incomplete. If it is incomplete, while some of the muscles return to their natural condition others are left paralyzed. The muscles thus left paralyzed then become atrophied. A permanent paralysis and atrophy is followed by deformities and arrest of development of the affected limbs.

There is no loss of sensation. The reflexes of the skin and muscles are diminished.

The Course of the Disease —The invasion of the symptoms is usually acute, less frequently subacute; the constitutional symptoms are absent, or present in varying degrees of severity. The paralysis is fully developed in a few hours, or not until after days or weeks.

The stationary period lasts for days, weeks, or months. It may be interrupted by fresh invasions of the disease with constitutional symptoms and paralysis of new groups of muscles.

The return of motor power in the paralyzed muscles is rapid or slow, occupying weeks or months. It is complete, all the muscles returning to their natural condition; or incomplete, some of the muscles recovering while others remain paralyzed for a long time, or permanently.

Prognosis.—The disease is seldom fatal. It is possible, however, for the patients to die with unusually severe constitutional symptoms, or with a paralysis involving the trunk as well as the extremities.

The prognosis as to the paralysis is determined by

examining the affected muscles with the faradic current. No loss of faradic irritability is followed by rapid recovery.

If there is no loss of faradic irritability until two or three weeks after the invasion, ultimate recovery is probable.

If there is loss of faradic irritability within ten days after the invasion, the muscles will become atrophied and remain paralyzed for a long time, or permanently.

Treatment.—During the active stage of the disease the patient is to be kept in bed, on a restricted diet, with mild counter-irritation over the spinal column. During the stationary period the counter-irritation may be continued, and the use of massage, passive motion, and electricity commenced. During the stage of improvement the patient is no longer to be kept in bed, is to be well fed, and the massage, passive motion, and electricity are to be continued.

If any muscles are left permanently paralyzed great care must be used for a long time to keep up the nutrition of the limb, and to prevent deformities by proper mechanical appliances.

AMYOTROPHIC LATERAL SCLEROSIS—CHRONIC SPINAL MUSCULAR ATROPHY—*Lesions.*—The disease involves the anterior gray cornua and the pyramidal tracts on both sides of the cord. In the pyramidal tracts there is degeneration of the nerve-fibres and hypertrophy of the connective tissue stroma; in the anterior gray cornua there is degeneration of the nerve-cells and fibres. The lesion begins in the cervical portion of the cord and extends downward, and later upward.

Causes.—The disease is more common in males than in females. It occurs in persons between the ages of twenty-five and forty-five years. There may be a previous history of syphilis or of exposure, or no cause may be found.

Symptoms.—The symptoms of the disease are developed slowly and gradually, and may become stationary at any time. They begin first in one arm and then extend to the other.

There are disturbances of sensation : formication, tingling, numbness, pains along the course of the nerves, and exaggerated tenderness of the muscles. There is gradual and incomplete loss of power in the muscles. The muscles become atrophied and may exhibit involuntary fibrillary contractions.

Then the loss of power gradually extends to the muscles of the legs, but without much atrophy. Sensation is preserved. In the atrophied muscles reflex action is lost, and the faradic and voltaic irritability are diminished. In the muscles which are not atrophied the reflex action is exaggerated.

As the disease advances the muscles become contracted and rigid ; at first temporary spasmodic rigidity, later a permanent contracture of both flexors and extensors, with occasional spasmodic action. The cases vary as to whether the atrophy of the muscles or their rigidity is the most developed.

Finally the muscles of the neck, the throat, and the tongue become involved, and the patient dies from starvation.

The course of the disease is a long one, protracted

over years, and it may at any time become stationary and not advance further for a long time.

LOCOMOTOR ATAXIA.—*Lesions.*—There is a degeneration of the nerve-fibres and a growth of new connective-tissue in the stroma, beginning in the lateral portion of the posterior columns, in the lower dorsal and lumbar regions. Opinions differ as to whether the degeneration of the nerve-fibres or the growth of new connective-tissue is the primary part of the lesion.

The disease, beginning in the lateral portions of the posterior columns, extends first to the posterior nerve-roots and the columns of Goll. Later it may involve the rest of the posterior and lateral portions of the cord, and even invade its anterior portions.

Similar changes are also developed in the optic nerves, the third, fifth, and sixth pair of nerves, and in the peripheral nerves.

Causes.—The disease is more common in males than in females. Most of the cases are developed between the ages of twenty and fifty years. In a considerable number of the patients there is a history of constitutional syphilis.

Symptoms.—The disease, if it runs its full course, runs through three stages, but very often it does not advance beyond the first or second stage.

The first stage is characterized by disturbances of sensation, pains, lessening of the reflexes, affections of the ocular muscles. The lessening of the muscular reflex is most readily determined by striking the tendon of the patella. This loss of reflex is almost constant, and occurs early in the disease.

The pains are severe and occur in paroxysms. They

may follow the course of the nerves, or occur in circumscribed regions. They may remain confined to a single nerve or a single region, or involve several nerves or several regions, or skip about from one place to another. They are most common in the legs, but may also occur in other parts of the body. The paroxysms vary in different cases as to their frequency, their severity, and their duration.

The Crises.—This name is given to attacks of pain and spasmodic muscular contraction occurring in the larynx, the stomach, the intestines, and the bladder.

In the larynx the attack of pain and spasmodic muscular contraction may assume one of two forms.

(1.) The patients have an attack of pain in the larynx, with a paroxysmal cough, like that of whooping-cough. Each paroxysm only lasts for a few seconds, but a number of such paroxysms are repeated at short intervals before the attack is finished.

(2.) The patients have attacks like those of laryngismus stridulus. There is a spasmodic contraction of the glottis, with urgent dyspnoea, which may last for a few minutes or for several hours. In the severe and protracted attacks the patients may lose consciousness or have general convulsions.

With the gastric crises there are pain and vomiting. The attacks may last for a few hours or for several days.

The intestinal crises are characterized by pain and diarrhoea.

The crises affecting the bladder are attended with frequent and painful micturition.

The disturbances of sensation. There is hyperæsthesia or anæsthesia of circumscribed portions of the skin. There is the feeling of constriction around the thorax, the abdomen, or the legs. There may be the sensations of numbness, pricking, and tingling, or as if something were interposed between the soles of the feet and the ground, or of muscular fatigue.

The affections of the ocular muscles. The iris and the ciliary muscle lose their capacity for reflex action and do not contract under the stimulus of light ; later they become paralyzed altogether.

There is either transitory or permanent paralysis of one or other of the recti muscles, with consequent diplopia or strabismus.

Additional symptoms. The patients may develop blindness of one or both eyes as the result of the changes in the optic nerves.

There may be retention or incontinence of urine.

There may be very obstinate constipation.

In males the sexual power is often diminished, or lost.

In a moderate number of cases there is disease of the joints—the knee, shoulder, elbow, hip, or wrist joints. There is a rapid swelling of the joint, due to the distention of the synovial sac with serum, but without heat or pain. This swelling may last for a time and then disappear without further trouble, or it may be followed by more serious changes. There is developed a chronic inflammation of the articular ends of the bones, with destruction of the cartilages, and rarefying osteitis.

The first stage of the disease lasts for months or years, and the disease may never advance beyond this first stage.

In the second stage of the disease the symptoms of the first stage continue, but there is added a new symptom—muscular ataxia.

The ataxia is developed slowly or rapidly. If it is developed rapidly it may be preceded by a febrile movement lasting for a few days.

The affected muscles retain their power, and are still, to a certain extent, under the control of the patient. But he cannot control the degree of contraction, nor can he co-ordinate the contraction of different groups of muscles. The muscles most frequently affected are those of the legs, but those of the arms may also be involved.

There is a great difference in the patients as to the degree of the development of the ataxia. It may never advance farther than to make walking somewhat difficult, or it may make the legs, and even the arms, almost useless.

The mental faculties may remain entirely normal ; or there may be loss of memory, emotional excitability, restlessness and sleeplessness, and inability for mental exertion.

The second stage of the disease lasts for years, and most of the patients never advance beyond this stage.

In the third stage the ataxia is succeeded by an entire loss of control of the affected muscles and paralysis is established.

The entire duration of the disease extends over many years. Its progress is slow, but the development of the ataxia may be rapid. There are often periods of intermission and improvement, and the disease may stop altogether at any time in its course.

The prognosis is not good, but yet there may be long periods of arrest and improvement, and the disease may never advance so far as to interfere with the usefulness of the patient.

Treatment.—During the first stage of the disease attention to the general health and mode of life, anti-syphilitic treatment, and hot douches or the actual cautery over the spine seem to be of service. The pains may be alleviated by rest, antipyrine, antifebrine, and opium. The laryngeal crises may be relieved by inhalations of the nitrite of amyl.

During the second stage the regulation of the mode of life is still of importance, the hot douches may be continued, the application of the galvanic, or faradic current to the affected muscles may perhaps be of service.

PERIPHERAL NEURITIS.

Lesions.—In persons who die while still suffering from this disease, one of two lesions are found. The affected nerves may be swollen, congested, infiltrated with serum and pus, the nerve-fibres degenerated; or there is simply degeneration of the nerve-fibres, with or without an hypertrophy of the interstitial connective-tissue.

The smaller nerve-trunks are the ones regularly involved.

Causes.—Chronic poisoning with lead, arsenic, or alcohol; syphilis; malaria; and the infectious diseases, especially diphtheria, seem to be real causes of this disease. The disease called beriberi seems to be an infectious disease, with peripheral neuritis as its charac-

teristic lesion. There are also a considerable number of cases for which no cause can be discovered.

Symptoms.—The Alcoholic Cases.—These are more common in females than in males. There are first the symptoms of the alcohol-poisoning—chronic gastritis, insomnia, general feebleness. Then disturbances of sensation—feelings of numbness and tingling; hyperæsthesia of the skin and muscles; pains along the course of the nerves, in the joints, the limbs, and other parts of the body; loss of tactile sensation; lessening of the reflexes. Then suddenly or gradually loss of motor power, first of the legs, later of the arms. The paralysis is especially marked in the extensor muscles. It is usually confined to the muscles of the arms and legs, but may involve those of the trunk also. The paralyzed muscles become soft, atrophied, and lose their faradic irritability.

In the severe cases there are also marked prostration and cerebral symptoms: loss of memory, mental feebleness, alternating delirium and stupor.

The severe cases get steadily worse and die—feeble, emaciated, and unconscious.

The milder cases run a course of many months. They get worse for a time, then remain stationary, and then gradually improve. The recovery is complete, or some of the muscles are left paralyzed and atrophied.

The Idiopathic Cases.—These are said to be more common in males than in females.

There are disturbances of sensation—numbness and tingling; hyperæsthesia of the skin and muscles; pains along the nerves, in the joints, in the limbs, and in other

parts of the body ; diminution of tactile sensation and of the reflexes.

The motor paralysis is developed rapidly or slowly. It begins in the muscles of the legs and then extends ; or it involves the muscles of the legs and arms at the same time. The muscles of the legs and forearms are the ones usually affected, less frequently those of the thighs, arms, and trunk. The paralyzed muscles become atrophied and lose their faradic irritability.

There may be cedema of the skin of the feet and hands, and around the joints. The joints themselves may become inflamed.

In the severe cases there are a febrile movement, marked prostration, and alternating stupor and delirium. These symptoms extend over months or years. Most of the cases recover, either completely, or with the paralysis of only a few muscles. But the severe cases may terminate fatally.

Treatment.—A previous history of lead-poisoning, or of syphilis, or of malaria gives an indication for the special treatment belonging to these diseases.

For the idiopathic cases the drugs most highly recommended are salicin, the salicylate of sodium and the bromides. The severe pains can only be relieved by morphine.

After improvement has begun, massage, passive motion, electricity, strychnia, and arsenic are of service.

ACUTE CATARRHAL INFLAMMATION OF THE PHARYNX, AND OF THE TONSILS.

Lesions.—In acute catarrhal pharyngitis the mucous membrane of the pharynx is congested, swollen, at first dry, later covered with mucus. Usually the mucus is in moderate quantity, but sometimes it covers most of the posterior wall of the pharynx.

In acute catarrhal tonsillitis one or both tonsils are congested and swollen, and their crypts are filled with mucus and desquamated epithelium, so that the surface of the congested tonsil is mottled with small white patches.

Causes.—Some persons, and the members of some families, are unusually liable to these inflammations. They are especially frequent during the winter months, and in cold and damp localities. They may be produced by the inhalation of irritating gases, or contaminated air.

Symptoms.—The symptoms are constitutional and local. The constitutional symptom is fever, with the accompanying prostration, vomiting, headache, and pains in the limbs. This often precedes the sore throat, and disappears before the latter has subsided.

The principal local symptom, besides the appearance of the throat, is pain, which is increased by any movement of the muscles of the pharynx.

The intensity of the fever and the severity of the pain vary in the different cases.

The inflammation is regularly a self-limited one, lasting for from four to seven days, but occasionally the cases are protracted for a longer period.

Treatment.—During the earlier stages of the disease the use of calomel, the sulphate of magnesia, sweating of the entire skin, aconite, and the salicylate of soda, either separately or combined in various ways, are of service. During the later stages, mild astringents applied by the spray, the brush, or used as gargles, give relief to the patient.

CROUPOUS TONSILLITIS.

Lesions.—Either one or both tonsils are congested, swollen, and covered with a layer of false membrane. The false membrane is composed of fibrine, pus-cells, epithelium in the condition of coagulation necrosis, and contains bacteria. The mucous membrane beneath is infiltrated with fibrine and pus-cells.

Causes.—Croupous tonsillitis occurs most frequently as one of the lesions of diphtheria; but it is also found as an independent local inflammation, and as a complication of the infectious diseases.

Symptoms.—A croupous tonsillitis, not belonging to diphtheria, gives the same symptoms as does a catarrhal tonsillitis, but in a more marked degree.

The febrile movement, with its accompanying symp-

toms, is more decided ; the appearance of the inflamed tonsils is different. The duration of the inflammation is only for a week, but the prostration may last for several days longer.

Treatment.—The cases may be managed in the same way as a catarrhal tonsillitis. But a more speedy cure may be effected by an hourly gargle of a solution of bichloride of mercury of the strength of 1 to 4,000.

SUPPURATIVE TONSILLITIS.

Lesions.—There is a suppurative inflammation beginning in the tissue of the tonsil, which goes on to the formation of an abscess. Either one or both tonsils may be involved. The suppurative tonsillitis may be preceded, or accompanied, by a catarrhal tonsillitis and pharyngitis.

Causes.—There is a decided predisposition in some persons and in some families to this form of tonsillitis. Exposure to cold and wet seems often to be an exciting cause.

Symptoms.—There is a febrile movement which precedes and accompanies the local inflammation, and which often subsides before the latter has run its course.

The inflamed tonsil becomes more and more swollen, it fills up the pharynx, pushes the soft palate forward, and forms a tumor in the neck. The mucous membrane of the pharynx is swollen, the throat and mouth are constantly filled with mucus and saliva.

There are great pain in the throat, inability to swallow, fear of suffocation, sleeplessness, and much general discomfort.

In a few cases respiration is interfered with by swelling of the uvula, or œdema of the glottis.

If only one tonsil is inflamed, the abscess regularly ruptures at the end of a week, the pus is discharged, and the patient rapidly recovers. If both tonsils are inflamed the disease may be prolonged for two weeks.

The patients almost uniformly recover, but it is possible for death to be produced by œdema of the glottis.

Treatment.—During the first twenty-four hours of the tonsillitis we may try to abort the inflammation by the use of calomel, sulphate of magnesia, salicylate of soda, and cold applied to the neck.

After the abscess has been formed, the inhalation of hot steam and the application of moist heat to the neck are of service. The excessive production of mucus can be partly controlled by astringent washes and the administration of belladonna. The pain and distress can only be alleviated by opium.

Swelling of the uvula and œdema of the glottis demand free scarification.

LARYNGISMUS STRIDULUS.

This name is given to two different forms of spasmodic contraction of the muscles of the larynx.

1. There is a spasmodic closure of the glottis, which is almost complete, lasts for a few seconds, and is then followed by a loud, stridulous inspiration.

This occurs in young children, usually under two years of age, who are badly nourished, or who suffer from rickets

It occurs in attacks, which are repeated after short or long intervals.

The child suddenly stops breathing, the face and lips become pale, or slightly livid, the appearance is almost that of a dead person. Then, after a few seconds, there is a long, stridulous inspiration, and the attack is over. Sometimes the attacks are accompanied by rigidity of the muscles, or by general convulsions, or by unconsciousness. Occasionally the attacks are fatal.

At the time of an attack, if it is an alarming one, we may give inhalations of nitrate of amyl, or pass a tube through the larynx.

To prevent the attacks the nutrition of the child is to be improved in every possible way.

2. There is a spasmodic partial closure of the glottis, attended with difficult and stridulous breathing, which lasts for several hours.

This occurs in young children of all kinds, whether their previous health has been good or not. There is a decided predisposition in families and in individuals. Some of the attacks are due to indigestion, for others no cause can be discovered.

The attacks usually begin in the night and last until the next day. The child wakes up in the night with laryngeal dyspnoea, which after a time is followed by more or less venous congestion of the skin. But there is no fever, and, except for the dyspnoea, the child does not look ill. Although the dyspnoea may appear alarming, it always eventually subsides, even if it is left to itself; but an emetic will cause it to disappear more rapidly. The best emetics are those which act the quickest: the

yellow sulphate of mercury, apomorphia, ipecac, and antimony are those ordinarily used.

ACUTE CATARRHAL LARYNGITIS.

Lesions.—The mucous membrane of the larynx is congested, and at first dry. The stage of dryness lasts for from twelve to forty-eight hours, and is then succeeded by an increased production of mucus. With the production of the mucus the congestion and swelling of the mucous membrane diminish. The inflammation may extend to the trachea and bronchi or to the pharynx. It occurs both in adults and children.

1. *In Adults. Causes.*—The inflammation occurs without discoverable cause, after exposure to cold, from the inhalation of smoke or steam, with syphilis, phthisis, measles, scarlatina, variola, erysipelas, typhus and typhoid fevers.

Symptoms.—In the more severe cases there is a febrile movement, in the milder cases this is absent.

The patients have a laryngeal cough, at first dry, afterward with mucous expectoration. The voice is husky, or stridulous, or reduced to a whisper. There is more or less discomfort or pain in the larynx. In some of the cases there is laryngeal dyspnoea, continuous, but with exacerbations. The most alarming feature about this dyspnoea is that occasionally the patients stop breathing suddenly and die.

The inflammation usually runs its course within two weeks, but it may be protracted for a much longer period.

Treatment.—During the acute stages of the laryngitis

the continuous application of hot sponges to the neck, and the inhalation of steam, are of decided service. The internal administration of tartarized antimony, or of the iodide of potash, may also be useful. When the dyspnoea is urgent the patients are to be constantly watched, in order that intubation or tracheotomy may be performed, if necessary. If the inflammation is prolonged the mineral acids, quinine, iron, and change of climate may hasten the subsidence of the disease.

2. *In Children.*—The catarrhal laryngitis of children is often called catarrhal croup. It is one of the most common diseases of childhood.

It occurs often without discoverable cause, sometimes after exposure to cold and wet, from the inhalation of steam and smoke, and as a complication of measles and scarlatina.

The disease is most common in children between the ages of one and five years, less frequent in older children. There is a well-marked predisposition to the disease in some children and in some families.

The changes in the mucous membrane of the larynx are the same as those observed in adults, but the smaller size of the larynx in children causes the swollen mucous membrane to be a still greater obstruction to the act of breathing.

Symptoms.—The local symptoms are: Dyspnoea, which is continuous, but with exacerbations; in some cases, attacks of laryngismus stridulus; stridulous voice, or loss of voice; and stridulous cough.

The general symptom is a rise of temperature with its accompanying disturbances. The fever may precede,

or follow, or be simultaneous with, the local symptoms.

The invasion of the disease is often sudden, and then usually occurs in the night, with the continuous dyspnœa, or an attack of laryngismus stridulus as the first symptom.

In other cases the invasion of the disease is gradual, with a croupy cough as the first symptom.

The ordinary cases last for three days and nights, the symptoms worse during the night and better during the day.

The patients vary as to the height of the temperature and the degree of the dyspnœa. Most of the patients are decidedly better by the fourth day, the improvement beginning with the production of mucus from the inflamed membranes. Occasionally, however, the laryngitis is protracted for one or two weeks, or it may be succeeded by a bronchitis.

The Prognosis is good, even the severe cases are seldom fatal.

Treatment.—For the laryngitis we apply moist heat to the neck, and give internally five- or ten-drop doses of the wine of antimony, every one or two hours. For the attacks of laryngismus stridulus we give emetics. For the fever it may be proper to use small doses of antifebrin or tincture of aconite; for the restlessness, small doses of opium. In the protracted cases small doses of calomel may be of service.

CROUPOUS LARYNGITIS.

MEMBRANOUS CROUP. *Lesions.*—The mucous membrane of the larynx is congested, swollen, and infiltrated with fibrine and pus. Its free surface is coated with a false membrane composed of fibrine, pus, and necrotic epithelium.

Causes.—Croupous laryngitis is most frequently seen as one of the lesions of diphtheria, but it may also be caused by inhalations of steam and smoke, by the poisons of the infectious diseases, and by exposure to cold and wet. It is much more common in children than in adults.

The Symptoms are the same as those of a catarrhal laryngitis, but are more severe, more continuous, and last for a longer time. The temperature is higher, the dyspnœa is more severe, and the evidences of imperfect aëration of the blood are more marked. In the favorable cases, after from four to seven days, the inflammation subsides, the false membrane becomes loosened, is coughed up, and the dyspnœa is relieved. In the unfavorable cases the dyspnœa continues, and causes the death of the patient.

The Prognosis is unfavorable, especially in children under three years of age.

The Treatment is the same as that for a catarrhal laryngitis, but in addition it is often necessary to employ intubation or tracheotomy to relieve the dyspnœa.

THE PHYSICAL SIGNS OF THE PLEURA AND OF THE LUNGS.

PERCUSSION.—When we percuss the wall of the chest we obtain certain sounds, and of these sounds we note the quality, the pitch, the intensity, and the duration.

1. *Pulmonary Resonance*.—This is characterized by pulmonary quality, low pitch, considerable duration, and variable intensity. It is heard over the healthy lung.

2. *Dulness*.—Of this the quality is imperfectly pulmonary, the pitch is higher, the duration is short, the intensity is small. It is heard over the normal lung where the chest-wall is thickened by bone or muscle, and where the liver and heart are in contact with the lung. It is heard over pleuritic adhesions, over small quantities of fluid in the pleural cavities, over consolidations of the lung, emphysema, enlargements of the heart, liver, and spleen, aneurisms of the aorta, abscesses, and tumors.

3. *Flatness*.—The quality is flat, the pitch is high, the duration is short, the intensity is small. It is heard over the liver, where it is uncovered by the lung, over thick pleuritic adhesions, fluid in the pleural cavities, complete consolidation of the lung, aneurisms, abscesses, tumors, and rarely over a lung which is not consolidated, but of which the bronchi are completely obstructed.

4. *Tympanitic Resonance*.—The quality is tympanitic, the pitch is high or low, the duration is considerable, the intensity is marked. It is heard over the stomach and intestines, over air in the pleural cavities, over cavities in the lungs, over solidified and compressed lung, and with emphysema.

5. *The Cracked Pot Sound*.—The quality is metallic, the pitch is high, the duration and intensity are not very great. It is heard over cavities, and over consolidated and compressed lung.

6. *Amphoric Resonance*.—The quality is amphoric, the pitch is low, the duration and intensity are considerable. It is heard over air in the pleural cavity, over large cavities in the lung, and occasionally over consolidations of the lower lobe of the left lung.

THE BREATHING.—In listening to the breathing we distinguish the sound of inspiration and that of expiration, and of each we note the quality, the pitch, the intensity, and the duration.

1. *Pulmonary, or Vesicular Breathing*.—Of inspiration, the quality is pulmonary, the pitch is low, the duration is considerable, the intensity is variable. Of expiration, the pitch is low and the duration is shorter.

2. *Bronchial Breathing*.—Of inspiration, the quality is bronchial, the pitch is high, the intensity and duration are variable. Of expiration, the pitch is higher and the duration is longer than those of inspiration. It is heard over consolidated and compressed lung, and over cavities.

3. *Broncho-vesicular Breathing*.—This is of a character intermediate between that of vesicular and that of bronchial breathing. It is heard over the normal lung in the interscapular region, and over the lesser degrees of consolidation and compression of the lung.

4. *Cavernous Breathing*.—Of the inspiration, the quality is cavernous, the pitch is low. The expiration is longer and lower pitched than the inspiration. It is

heard over cavities and over consolidated or compressed lung.

5. *Amphoric Breathing*.—The quality is amphoric, the pitch is low, the expiration is prolonged. It is heard over large cavities in the lungs, and over pleural cavities which contain air, with perforation of the lung.

6. *Sibilant Breathing*.—The quality is sibilant, the pitch is high, the intensity is great, the expiration is prolonged.

Sonorous Breathing.—The quality is sonorous, the pitch is low, the intensity is great, the expiration is prolonged. Both these forms of breathing are produced by a contraction of the calibre of the larger and medium sized bronchi. The ordinary causes of such a contraction are bronchitis and spasmodic asthma.

A special form of sibilant and sonorous breathing is produced by narrowing of the lumen of the trachea, or of the large bronchi.

RÂLES.—These are abnormal sounds which accompany the breathing and are not heard over the healthy lung.

1. *The Crepitant Râle*.—This is a very fine, dry, crackling sound. It is heard at the end of inspiration, is produced in puffs, and seems to be close to the ear. It is heard with dry pleurisy, with pneumonia, and with phthisis.

2. *The Subcrepitant Râle*.—This is a fine, moist sound; heard with inspiration, or with expiration, or with both. It accompanies dry pleurisy, bronchitis, pneumonia, phthisis, and cedema of the lungs.

3. *The Coarse, or Mucous, Râles*.—These are loud,

moist sounds, heard both with inspiration and expiration. They are heard with pleurisy, with bronchitis, with pneumonia, and with phthisis.

4. *The Gurgling Râles*.—These are loud, coarse, moist sounds, of gurgling quality. They are heard over small cavities, over compressed lung, and occasionally over pleuritic adhesions.

FRICTION SOUNDS.—These are produced by the rubbing of opposed surfaces of pleura coated with fibrine, or by the movement of pleuritic adhesions.

They have the characters of the crepîtant, the subcrepîtant, the mucous, or the gurgling râle ; the sound is a grazing, or rubbing, or creaking one.

THE VOICE. *The Pulmonary Voice*.—The quality is pulmonary, the pitch is low, the intensity and the thrill differ with the individual.

Increased Vocal Resonance.—The quality is pulmonary, the pitch is higher, the intensity and the thrill are greater. It is heard over the normal lung in the right infra-clavicular and in both the interscapular regions. It is also heard over consolidated or compressed lung, over cavities, and over lung which is adherent to the wall of the chest.

Diminished Vocal Resonance.—The intensity and the thrill are diminished, the quality and the pitch are the same as with the pulmonary voice. It is heard over small effusions in the pleural cavities, over thick pleuritic adhesions, over consolidated lung, and over lungs of which the bronchi are occluded.

Suppressed Vocal Resonance.—There is absence of the voice-sound with fluid in the pleural cavities, with intra-

thoracic tumors, with consolidation of the lung, and with obstructions of the bronchi.

Bronchophony.—The quality is bronchial, the pitch is high, the intensity and the thrill are variable. It is heard over consolidated and compressed lung, and over cavities.

Ægophony.—This is a form of bronchophony characterized by its peculiar, shrill quality. It is heard over lung compressed by fluid in the pleural cavity, at the level of the fluid.

Pectorilology.—Not only the sound of the voice is heard, but the articulation of words can be distinguished. It is heard over large cavities, and sometimes over consolidated lung.

THE PLEURA.

THE pleura is a connective-tissue membrane composed of fibrillated connective tissue, with its basement substance and cells, and covered over its free surface by a layer of endothelial cells. Imbedded in it are nerves, blood-vessels, and lymphatics. According to its distribution, we speak of the pulmonary, costal, mediastinal, and diaphragmatic pleura.

The pleura may become inflamed in several different ways, and we describe :

Pleurisy with the production of fibrine.

Pleurisy with the production of fibrine and serum.

Pleurisy with the production of fibrine, serum, and pus.

Pleurisy with adhesions.

Tubercular pleurisy.

In pleurisy with the production of fibrine alone, or of both fibrine and serum, the morbid changes in the pleura are essentially the same, differing only in the presence or absence of the serum.

The first change in the pleura is simply a congestion and a falling off of the endothelial cells. Then the

pleura loses its smooth, shining appearance and looks dull and rough, this change being due to the presence of small knobs and threads of coagulated fibrine on its surface. At the same time, if serum is to be present, it begins to collect in the pleural cavity.

Next there is a swelling of, and a new-growth from, the connective-tissue cells of the pleura, and an emigration of white blood-cells from the vessels. Then we find the pleura coated with a layer of fibrine in which are entangled pus-cells and new connective-tissue cells. After this the new connective-tissue cells entangled in the fibrine become more numerous, a basement substance and new blood-vessels are formed.

Finally, the fibrine and the serum are absorbed and disappear, and the pleura is left thickened and with connective-tissue adhesions binding together its opposed surfaces.

PLEURISY WITH THE EXUDATION OF FIBRINE. DRY PLEURISY.

Lesions.—The inflammation begins in the costal or pulmonary pleura, according to the cause producing it. It extends regularly to the portion of pleura opposite to it. Usually only a circumscribed portion of the costal, pulmonary, mediastinal, or diaphragmatic pleura is involved, but sometimes the entire pleura of one side of the chest is inflamed. The inflamed pleura is coated with fibrine, and bands of fibrine form adhesions between the opposed pleural surfaces.

In rare cases the quantity of fibrine is so great as to compress the lung.

When the inflammation has subsided, the fibrine is absorbed, but permanent connective-tissue thickening and adhesions are left.

Causes.—Exposure to cold and wet, wounds of the chest-walls, inflammations of the lung, peritonitis, the infectious diseases, and Bright's disease are regular causes of pleurisy. In some cases there seems to be an individual predisposition to pleurisy, and the same person suffers from several attacks.

Symptoms.—The most constant physical sign is a friction sound—a crepitant, subcrepitant, or mucous râle, or a rubbing sound. This is heard over the inflamed portion of the pleura. It cannot be heard if only the mediastinal or diaphragmatic pleura are inflamed. It is heard only with inspiration, or with expiration also. It is usually not continuous, but requires a forced inspiration to develop it.

In some cases there is also dulness over the inflamed portion of the pleura.

In the mild cases the only constitutional symptom is pain over the inflamed pleura.

In the more severe cases there is a febrile movement attended with prostration and headache, shallow breathing, and a dry cough. These symptoms only last for a few days.

The exceptionally severe cases, with very large exudations of fibrine, resemble cases of lobar pneumonia.

The ordinary cases recover after a short time, but the patient is left with permanent thickenings and adhesions of the pleura. Such adhesions may give no further trouble, except for occasional pain; or they may form the

starting-point for a chronic pleurisy with adhesions, followed by interstitial pneumonia and chronic bronchitis.

Treatment.—For the pleurisy we employ wet, or dry, cups, or blisters over the inflamed pleura. For the pain we use opium. While the febrile movement is present the patients should be kept in bed. So long as the friction sound persists the patient must be kept in the house if the weather is cold, but in warm weather this is not necessary.

PLEURISY WITH THE EXUDATION OF FIBRINE AND SERUM. PLEURISY WITH EFFUSION.

The inflammation involves the greater part of the pleura on one side of the chest. Occasionally both sides of the chest are inflamed, and when this is the case the pericardium is apt also to be inflamed. The pleural cavity contains more or less clear, or turbid, serum. The surface of the pleura is coated with fibrine, and bands of fibrine join together its opposed surfaces. The lung is more or less compressed, according to the quantity of fluid.

After the inflammation has subsided the serum and fibrine are absorbed, and thickenings and adhesions of connective tissue are left.

The compressed lung expands partially or completely. According to the expansion of the lung, there is left more or less retraction of the affected side of the chest.

The Causes are the same as those which produce dry pleurisy.

Symptoms.—The disease may run an acute, or a sub-acute course.

1. *The acute form.* The symptoms begin abruptly with chills, fever, full and frequent pulse, pains in the head and limbs, vomiting, and prostration. The breathing is frequent and shallow, there may be a dry cough, there is severe pain. The pain is referred to the inflamed pleura, or to some point in the back, or in the abdomen, or even to the opposite side of the chest. It usually becomes less severe with the accumulation of serum in the pleural cavity.

After a few days the acute symptoms subside. The inflammatory products remain in the chest for some time longer. They may then be absorbed, or the pleurisy may take on the subacute form and last for a long time.

2. *The subacute form.*—The symptoms are developed gradually and slowly. The patients complain of pain in the side, of dyspnoea on exertion, of a dry cough, of loss of appetite, flesh, and strength, and they become anæmic. They are, for a time, not confined to bed, and often continue at their work. They have a little fever, the temperature normal in the morning, but running up to 100° in the afternoon.

In some cases, however, the temperature runs higher: 100° in the morning and 101° to 103° in the afternoon. With these higher temperatures the patients lose flesh and strength more rapidly, and may sweat at night.

The physical signs.—At the beginning of the inflammation, when the pleura is coated with fibrine and but little serum has been exuded, there is a friction sound,

which is a rubbing sound, or a crepitant or subcrepitant râle. After the fluid has been absorbed there is again a friction sound—a subcrepitant or coarse râle, or a creaking sound.

When a considerable quantity of fluid has accumulated in the pleural cavity there are physical signs due to the presence of the fluid and the compression of the lung.

Below the level of the fluid there is flatness on percussion, absence of voice, of breathing, and of vocal fremitus. The fluid accumulates in the lower part of the pleural cavity, compressing the lung upward and against the vertebral column; or in the posterior part of the pleural cavity, compressing the lung against the anterior wall of the chest; or it may be shut in by adhesions. The compression of the lung is in proportion to the quantity of the fluid.

At the level of the fluid there is dulness on percussion and œgophony.

Above the level of the fluid, over the compressed lung, the percussion resonance is pulmonary, or exaggerated and high-pitched, or tympanitic. The breathing is pulmonary, or exaggerated, or broncho-vesicular, or bronchial.

The affected side measures more than the opposite side of the chest, the diaphragm is pushed down, the intercostal spaces may be forced outward, the heart may be displaced toward the opposite side of the chest.

As the fluid is absorbed the voice and breathing can be heard lower and lower down, the flatness disappears,

but dulness on percussion remains for some time after all the fluid has been absorbed.

Exceptional physical signs.—The vocal fremitus may not be lost below the level of the fluid. Bronchophony alone, or both bronchial voice and breathing, may be heard below the level of the fluid, especially if the quantity of fluid is large and the lung much compressed.

Above the level of the fluid, over the compressed lung, there may be cavernous breathing and gurgling râles.

The sacculated effusions give very irregular physical signs, varying with the position of the fluid.

The acute cases of pleurisy with effusion may last for only a few weeks, but more frequently, whether acute or subacute, they last for weeks or months.

In a few cases the disease terminates fatally. Death is then often sudden, and seems to be due to congestion and œdema of the other lung, or to interference with the action of the heart.

In a moderate number of cases the inflammation changes its character, pus is added to the other inflammatory products, and the patients have empyema.

Most of the patients recover, but with a damaged pleura, and with a lung which expands more or less incompletely. In many of them the only subsequent inconvenience is some pain on the affected side of the chest; but in others there is marked retraction of the wall of the chest, chronic pleurisy with adhesions, interstitial pneumonia, or chronic bronchitis. It may also happen that such a pleurisy will be succeeded by chronic phthisis.

The displaced heart may return to its natural position, or it may remain fastened in its new place by adhesions, or it may be drawn over to the retracted side of the chest.

Diagnosis.—Pleurisy with effusion may be mistaken for empyema, tubercular pleuⁱisy, pneumonia, phthisis, abscess of the liver, or tumors of the pleura. In many cases we are warranted in drawing off fluid from the pleural cavity with a fine needle, in order to establish the diagnosis.

Treatment.—Of the acute form of pleurisy with effusion, the treatment is that of an acute exudative inflammation. Wet or dry cups, or blisters over the affected side of the chest, calomel, and the sulphate of magnesia given internally, are the most efficient remedies. The patients are to be kept in bed and on a fluid diet. The pain and restlessness may be relieved by opium combined with aconite or veratrum viride, or by chloral hydrate with one of the bromides.

In subacute pleurisy with effusion we have to treat the inflammation and the accumulation of fluid within the pleural cavity. The only direct means of treating the inflammation is the use of counter-irritation over the affected side of the chest. The indirect means are : keeping the patients in bed, or getting them out of doors in a suitable climate, according to the case ; the use of iron, quinine, and the mineral acids ; and the regulation of the diet.

If the quantity of fluid in the pleural cavity is moderate it can be removed by diuretics : the iodide or acetate of potash, the diuretic pill, chloride of sodium,

caffein, convallaria, digitalis. At the same time the urine is to be measured every day, and the ingestion of fluids somewhat restricted.

If the chest is distended with fluid this must be removed by the aspirator. In doing this the strictest cleanliness must be observed, and only a moderate quantity of the serum withdrawn. Immediately after the aspiration the use of diuretics should be begun.

PLEURISY WITH THE PRODUCTION OF SERUM, FIBRINE, AND PUS. EMPYEMA.

Lesions.—The inflammation regularly involves the whole of the pleura on one side of the chest, less frequently a circumscribed portion of the pleura. The inflammation follows two different forms :

1. The pleura is coated with fibrine and pus, and its cavity contains purulent serum, but the pleura itself is but little changed. This form is most common in children.

2. The pleura is coated with fibrine and pus, its cavity contains purulent serum, and in addition the pleura itself is much changed. It is split up by great numbers of new cells, so that it resembles granulation tissue.

In either case the fluid accumulates in the lower part of the pleural cavity, pushing the lung upward and toward the vertebral column ; or in the posterior part of the pleural cavity, pushing the lung forward ; or it is sacculated in any part of the pleural cavity. The lung is usually much compressed. In old cases the pleura becomes much thickened, and may be infiltrated with the salts of lime.

The suppurative process may extend from the pulmonary pleura to the lung, and the pus then escape at intervals from the bronchi ; or it may extend from the costal pleura to the wall of the chest, and the pus escape externally.

In a few cases the inflammatory products and the superficial layers of the pleura become gangrenous.

Symptoms.—1. The inflammation may be primary, after exposure to cold, or without discoverable cause. The patients are suddenly attacked with chills, a high temperature, marked prostration, headache, pains in the back and limbs, pain over the inflamed pleura, shallow and painful breathing, sometimes cough. The symptoms may continue acutely and the patients die in a short time, or they may subside and the inflammation pass into the chronic condition.

2. The inflammation may be secondary to a pleurisy with effusion, or to a lobar pneumonia. A pleurisy with effusion may change suddenly or slowly into an empyema. The patients lose flesh and strength more rapidly, and have higher temperatures. A lobar pneumonia may run its course, convalescence be established and continue for several days, and then the temperature goes up and there are the physical signs of fluid in the pleural cavity.

3. An empyema, after running its course for a shorter or longer time, will suddenly change, the inflammatory products become gangrenous, the patients pass into the pyæmic condition and die in a few days.

4. Abscesses in the wall of the thorax, in the liver, in the abdominal cavity, or in the lung may rupture into the pleural cavity and set up a purulent inflammation.

The physical signs of empyema are the same as those of pleurisy with effusion, but sacculaton of the fluid and irregular physical signs are more common.

The course of the disease.—Some of the acute cases continue without any abatement of the symptoms, and terminate fatally within a short time. More frequently the course of the disease is chronic. The patients go on for months or years with fever, gradual loss of flesh and strength, and dyspnœa and cough. In some the lung is perforated and the pus from time to time coughed up through the bronchi; in some the wall of the chest is perforated and the pus imperfectly evacuated; in some there is septic poisoning.

Very rarely does spontaneous recovery take place; somewhat more frequently there is partial recovery, with absorption of some of the pus and sacculaton of the remainder. Most of the patients, if not cured by proper treatment, die exhausted by the disease, or with pulmonary phthisis, or with waxy viscera.

The prognosis is more favorable in children than in adults, in those operated on early than in those operated on late. It is unfavorable after septic poisoning has begun, and when the empyema is caused by the rupture of an abscess into the pleural cavity.

The diagnosis is between empyema, pleurisy with effusion, lobar pneumonia, pulmonary phthisis, and abscess of the liver.

The treatment.—In a very few cases, especially in children, empyema can be cured by repeated aspirations, but as a rule it is necessary to make an opening in the wall of the chest. The opening is usually to be made as

low down as possible, just in front of the axillary line, and in many cases portions of one or more ribs must be resected. It is often wise to draw off part of the fluid with the aspirator one or two days before the operation is performed. It may be proper to introduce the hand into the pleural cavity, break up the adhesions between the lung and the wall of the chest, and sweep away the masses of fibrine which coat the pleura. The operation is to be done with the strictest antiseptic precautions. After the operation the antiseptic dressings should be changed as seldom as possible, no attempt should be made to wash out the tube or the chest, and, if the case is to do well, the drainage-tube should be removed by the end of the fourth week. It is better to operate a second time and to remove more of the ribs, than to leave the drainage-tube in the chest for too long a time.

CHRONIC PLEURISY WITH ADHESIONS.

Lesions.—There is a chronic inflammation of the pleura with the production of new connective tissue, but without fibrine, serum, or pus. The inflammation begins at some part of the pleura, and then extends until first one lung and then both are completely covered with adhesions and fastened to the walls of the chest. It is not to be confounded with the old adhesions found over so many lungs after death, but is a chronic inflammatory process with the progressive formation of more and more adhesions.

Causes.—The disease usually originates in adhesions which have been left behind by previous attacks of dry pleurisy, pleurisy with effusion, or pneumonia; but in

some cases no history of such previous acute attacks can be obtained.

The symptoms vary with the extent of the lesions.

In the early stages the only symptoms are occasional pain over the affected part of the chest, dulness on percussion, and friction sounds.

When the disease is farther advanced the pain continues, there is a dry cough, the breathing is imperfect, there is dyspnoea on exertion, and the area of dulness on percussion and of the friction sounds is larger.

In the advanced cases the difficulty in breathing becomes very marked, the cough is more troublesome, the heart is diminished in size and sometimes displaced, the circulation is feeble, the patients lose flesh and strength. They usually die from some intercurrent disease, but occasionally the pleurisy is the only discoverable cause of death.

Treatment.—The patients should live as much as possible in the open air. They may be benefited by the use of cod-liver oil, iron, quinine, or the mineral acids. They should practise daily the filling and emptying of the lungs with air in as complete a manner as is possible.

TUBERCULAR PLEURISY.

Apart from the tubercular inflammation of the pleura, which accompanies general tuberculosis and chronic phthisis, we find tubercular pleurisy occurring as a localized tubercular inflammation.

Lesions.—The inflammation involves regularly the whole of the pleura on one side of the chest, the costal

pleura being principally involved. The pleura is of a bright-red color mottled with small white points, or is only thickened and coated with fibrine. The tissue of the pleura is split up by the growth of new connective-tissue cells, and contains numerous tubercle granula. There is a large quantity of fluid in the pleural cavity, which is blood-stained, or purulent, or clear.

Symptoms.—The clinical history is that of pleurisy with effusion, or of empyema, but the patients do badly. They lose flesh and strength, the fluid accumulates rapidly after it has been drawn off, the inflammation of the pleura persists, and the patients die, either suddenly or exhausted by the disease, within a few weeks or months.

HYDRO-PNEUMOTHORAX.

This name is used to designate the presence of both air and fluid in the pleural cavity. Such a condition may be established in several different ways.

There may be a gangrenous empyema with the formation of gas in the pleural cavity.

There may be an empyema with an opening through the wall of the chest or into the lung.

There may be abscess, or gangrene of the lung perforating the pulmonary pleura.

There may be pulmonary phthisis with softening of cheesy nodules and perforation of the pleura.

Symptoms.—The only cases of hydro-pneumothorax which have a special clinical history are those due to the rupture of abscesses or phthisical nodules in the lung. There is first the history of the previous lung disease. Then, suddenly, at the time of the perforation,

there is severe pain, a feeling as if something had given way within the chest, urgent dyspnoea, a rapid and feeble heart action, and great prostration. The patients may die in collapse within a few hours of the commencement of the attack ; or the urgent symptoms may subside and the patients continue to live for some time with the symptoms of empyema and phthisis.

Physical signs.—The affected side of the chest is larger than the other, and moves but little with respiration. The heart and the diaphragm are displaced. Vocal fremitus is absent. Percussion gives, above the level of the fluid, exaggerated pulmonary or tympanitic resonance, or flatness ; below the level of the fluid, flatness. Auscultation gives, above the fluid amphoric breathing, or absence of breathing ; below the level of the fluid, absence of breathing. If the patient is shaken we get the splashing sound of the fluid in the chest called “succussion.” We may also get the sound resembling drops of liquid falling into liquid, called “the metallic tinkle.”

THE LUNGS.

THE lungs first appear as two small protrusions on the front of the œsophagus. They are diverticula of the hypoblast surrounded by mesoblast. The formative process consists in the budding of hypoblastic into mesoblastic substance ; the hypoblast furnishing the lining epithelium and the mesoblast the stroma. As the lungs continue to develop they look like glands with acini and ducts.

Just before birth the lungs are fully formed, but the air-spaces are not dilated and are completely lined with cuboidal epithelium. After birth the air-vesicles are dilated and their epithelium is flattened.

As the child becomes older the air-spaces occupy a larger, and the bronchi and the stroma a smaller, part of the lung.

In the adult lung the stroma is arranged so as to divide the lung up into lobules, six- or seven-sided blocks, each side 5 to 15 mm. long. But few of the lobules are entirely separated from each other by the stroma. The small bronchi enter the lobules irregularly and break up into terminal bronchioles. The bronchioles terminate in the air-passages. The air-vesicles are given off from the air-passages or directly from the bronchioles. The

walls of the air-passages and vesicles are thin, connective-tissue membranes with scattered flat cells on their surfaces.

BRONCHITIS.

Causes.—Inflammation of the bronchi occurs at all ages; but its acute form is most common in children, and its chronic form most common in old persons.

Persons living in cities, those who are much confined to the house, and those whose health has been enfeebled, are liable to the disease. There is in some persons an individual predisposition to inflammations of the bronchi. The disease is especially prevalent in cold and damp climates and during the cold and wet months of the year. As exciting causes of bronchitis we recognize exposure to cold, the inhalation of irritating gases and substances, and (probably) pathogenic bacteria. Measles, whooping-cough, and many of the infectious diseases are often complicated by bronchitis.

1. *The acute catarrhal bronchitis of adults.*

Lesions.—The inflammation involves the trachea, the large bronchi, and the medium-sized bronchi, not often the smaller ones. As a rule, the bronchi of both lungs are equally inflamed. The mucous membrane of the bronchi becomes congested and swollen, at first dry, afterward coated by an increased production of mucus. There are also desquamation of the epithelial cells, a moderate emigration of white blood-cells, sometimes diapedesis of the red blood-cells. The lumen of the bronchi may be narrowed, either by the congestion and swelling of the mucous membrane or the contraction of the muscular coat. *

Symptoms.—The mild cases of acute bronchitis are attended with cough accompanied by scanty mucous sputa, by pain over the sternum, and a moderate feeling of indisposition. There may be no physical signs, or a few coarse râles, or sibilant and sonorous breathing. The natural period of the inflammation seems to be about one week, but it is often prolonged for a much longer time.

In the more severe cases there is cough with mucous, or blood-streaked, or muco-purulent sputa. There is a febrile movement which precedes and accompanies the local symptoms. There may be a good deal of bronchial dyspnoea. There are coarse râles heard usually over both lungs, sometimes only over one, and in some cases sibilant and sonorous breathing. The cases vary a good deal as to their severity, but their natural termination is in recovery after one or two weeks. In some of the cases a localized broncho-pneumonia is developed, with dulness and increased vocal resonance over the consolidated portion of lung.

There are cases in which the bronchitis, instead of running its natural course, continues. The patients have cough with muco-purulent sputa, an irregular fever, higher in the evening, night-sweats, loss of flesh and strength. These symptoms may continue for weeks or months.

Treatment.—The milder cases are not confined to bed, but are to be cautioned against undue exposure. Many of them recover without any treatment, but in some it is necessary to use counter-irritation over the chest, and to give combinations of ipecac, belladonna, opium, and quinine in small doses frequently repeated.

In the more severe cases the patients are in bed. The counter-irritation over the chest must be employed more energetically. It may be necessary to give calomel or sulphate of magnesia ; or the use of ipecac, aconite, veratrum viride, or opium may be indicated. If the dyspnoea is a marked feature, we should endeavor to make out whether it is due to the congestion of the bronchi or to the contraction of their muscular coats. If it is due to congestion, the drugs which increase the production of mucus are indicated : pilocarpine, quebracho, grindelia robusta, ipecac. If it is due to contraction of the muscular coat, we employ the drugs which relax muscular spasm : nitrite of amyl, nitro-glycerine, chloral hydrate, opium.

In the protracted cases the most efficient treatment is to send the patients to a different climate. If this cannot be done we employ quinine, iron, the mineral acids, turpentine, strychnia, oxygen.

2. *The acute catarrhal bronchitis of old persons.*

The lesions are the same as in the bronchitis of adults.

The symptoms are more severe in proportion to the extent of the bronchitis, and the disease is not as well borne as it is in adults. There are, from the first, much prostration ; an irregular febrile movement ; a rapid and feeble pulse ; difficult and oppressed breathing ; cough with mucous expectoration ; restlessness, sleeplessness, sometimes delirium ; loss of appetite, nausea, vomiting. There are coarse and subcrepitant râles over both lungs, or only over a portion of one lung. In some cases no râles can be heard.

An acute bronchitis in old persons is often alarming and sometimes fatal.

The treatment is the same as in adults ; but the nursing is even more important, the difficulty in feeding the patients is greater, and stimulants are more likely to be necessary.

3. *The acute catarrhal bronchitis of children.*

The lesions are the same as in adults : congestion and swelling of the mucous membrane, after a time an increased production of mucus with some subsidence of the swelling. But as the lungs of children are smaller, as the bronchi constitute a relatively larger portion of the lung, as the bronchi are smaller and more easily occluded, as such an occlusion may even be complete with unaëration of portions of the lung, so we find bronchitis in children to interfere seriously with the function of breathing and to constitute a serious disease.

Symptoms.—An acute bronchitis may be preceded by coryza, pharyngitis, tonsillitis, or laryngitis ; it may occur as a primary inflammation ; or it may complicate measles, whooping-cough, or one of the infectious diseases.

In the mild cases there is no fever, the children hardly feel sick ; but they cough, and coarse râles can be heard over both lungs. The inflammation regularly runs its course and subsides within one or two weeks.

In the majority of these mild cases no treatment is necessary, not even for the cough. It is wise, however, to keep these patients in the house until the bronchitis has subsided, unless the weather is warm and good.

In the more severe cases the invasion of the inflammation is marked in some cases by general convulsions. There is a well-marked febrile movement, the temperature higher in the afternoon, sometimes falling even to the normal in the morning. The pulse is rapid, but usually not feeble. The breathing is rapid, sometimes insufficient ; it may be made worse for a time by distention of the stomach with food. There may be alternating restlessness and drowsiness. There are subcrepitant and coarse râles heard over both lungs, sometimes early, sometimes late in the disease.

The inflammation regularly runs its course within two weeks, and the patients recover. But they are often alarmingly ill for several days, and may die from the disease.

In infants only a few weeks old, rapid breathing, fever, and prostration are the only symptoms, and the disease proves fatal within a few days.

Treatment.—During the first days of the inflammation we employ counter-irritation over the chest by turpentine, croton-oil, or poultices, and give small doses of calomel or sulphate of magnesia. Later ipecac, aconite, and opium may be of service. But it is to be remembered that in young children all drugs may do harm, and that too little treatment is better than too much.

4. *Chronic Catarrhal Bronchitis.*

Chronic catarrhal bronchitis may from the first have the character of a chronic inflammation ; it may follow one or more attacks of acute bronchitis ; it may complicate gout, emphysema, chronic endocarditis, interstitial

pneumonia, pleuritic adhesions, phthisis ; it may be produced by the inhalation of irritating substances.

Lesions.—The mucous glands produce too much mucus, they may be hypertrophied ; the walls of the bronchi are thickened or thinned ; the lumen of the bronchi may be narrowed or dilated.

Symptoms.—In the mild cases the patients are only troubled by the cough and expectoration, while their general health remains good.

In the severe cases the cough is more troublesome, the expectoration more profuse ; there may be constant or spasmodic dyspnœa ; there is an irregular fever and loss of flesh and strength. There are coarse and subcrepitant râles, sometimes sibilant and sonorous breathing.

The disease runs a protracted course, better in the summer and worse in the winter. The patients are more likely to die from some intercurrent disease than from the bronchitis.

Treatment.—The most efficient treatment is the selection of a proper climate—preferably a dry, inland, elevated region, either warm or cold.

As regards drugs, the mineral acids, iodide of potash, the preparations of turpentine, ipecac, and belladonna are sometimes of service.

5. *Acute Croupous Bronchitis.*

Lesions.—The inflammation involves the trachea and bronchi. The mucous membrane is congested, swollen, and coated with a layer of coagulated fibrine in which are entangled pus and epithelial cells. The smaller bronchi are not merely coated, but filled with the inflammatory products.

Causes.—Croupous bronchitis occurs with lobar pneumonia and diphtheria, from the inhalation of hot steam and smoke, and as a primary inflammation. It is possible that in some of the primary cases the patients really have diphtheria, but without inflammation of the throat or larynx.

Symptoms.—In the primary cases the disease may be preceded by a catarrhal tracheo-bronchitis.

The patients have chills, well-marked fever, prostration, very urgent dyspnoea, and cough. The sputa are at first mucous; later, mixed with fragments of false membrane. The patients develop the evidences of imperfect aëration of the blood—cyanosis, delirium, stupor. Over the lungs there are coarse râles, or absence of breathing. About half the patients die with dyspnoea within from three to fourteen days.

The treatment is the same as for a severe catarrhal bronchitis, but it is to be energetically used, and the constant inhalation of oxygen gas is of service.

6. *Chronic Croupous Bronchitis.*

There are patients who give the history and the symptoms of a chronic bronchitis, or of chronic phthisis, but who from time to time cough up casts of the bronchi, often of considerable size.

These casts represent bronchi of some size, with their branches. They are of white color and are composed of an albuminoid substance allied to fibrine or mucus.

Except for the occasional production of these casts there seems to be nothing to distinguish these cases from the ordinary cases of chronic bronchitis, or phthisis.

PNEUMONIA.

The inflammations of the lung, as distinguished from those of the bronchi and pleura, are called "pneumonia."

It is convenient, in describing the different varieties of pneumonia, to classify them in a somewhat arbitrary way, as follows :

Primary Lobar Pneumonia.

Secondary and Complicating Lobar Pneumonia.

Pneumonia of Heart Disease.

Broncho-pneumonia.

Interstitial Pneumonia.

Tubercular Pneumonia.

Syphilitic Pneumonia.

I. PRIMARY LOBAR PNEUMONIA.

The inflammation regularly involves the whole of one lobe, or the whole of one lung, or portions of both lungs.

Juergensen, from a study of 6,666 cases, gives the following table to show the relative frequency of the situation of the lesion :

	Per cent.
Right Lung.....	53.70
" " upper lobe.....	12.15
" " middle lobe.....	1.77
" " lower lobe.....	22.14
" " whole lung.....	9.35
Left Lung.....	38.23
" " upper lobe.....	6.96
" " lower lobe.....	22.73
" " whole lung.....	8.54

	Per cent.¹
Both Lungs.....	8.07
“ “ both upper lobes.....	1.09
“ “ both lower lobes.....	3.34

The inflammation in acute lobar pneumonia is of pure exudative type, characterized by congestion, emigration of white blood-cells, diapedesis of red blood-cells, and exudation of blood-plasma, while the tissue of the lung remains unchanged. For clinical purposes it is important to have as distinct an idea as possible of the condition of the lung while it is the seat of such an exudative inflammation, so that we describe the condition in which the lung is found while the inflammation is going through its regular stages of congestion, exudation, and resolution.

During the first hours of the inflammation only irregular portions of the lobe which is to be inflamed are involved; later, the entire lobe. The lung is congested, œdematous, tough, but not consolidated. The air-spaces contain granular matter, fibrine, pus-cells, red blood-cells, and epithelial cells. The epithelium remaining on the walls of the air-spaces is swollen; there are large numbers of white blood-cells in the capillaries. The larger bronchi are congested, dry, or coated with mucus; the small bronchi contain the same inflammatory products as do the air-spaces. The pulmonary pleura, as a rule, is not coated with fibrine. This is called the stage of "congestion." The stage of congestion regularly only lasts a few hours, but it may be protracted for several days.

When the exudation of the inflammatory products has

reached its full development, the presence of these products within the air-spaces and bronchi causes the lung to be solid, and at this time the lung is said to be in the condition of "red hepatization." The lung is now consolidated, red, its cut section looks granular, the granules corresponding to the plugs of inflammatory matter within the air-spaces. For some time after death the inflammatory products remain solid, and the cut section of the lung dry, but later, with the commencement of post-mortem changes, these products soften and the cut section is covered with a grumous fluid. The air-vesicles, the air-passages, the small bronchi, and sometimes the large bronchi, are filled and distended with fibrine, pus-cells, red blood-cells, and epithelium. In spite of the pressure on the walls of the air-spaces, the blood-vessels in their walls remain pervious. The pulmonary pleura is coated with fibrine and the interstitial connective tissue of the lung is infiltrated with fibrine. The hepatized lobe is increased in size, sometimes so much so as to compress the rest of the lung. About one-fourth of the fatal cases die in the stage of red hepatization, at any time from twenty-four hours to eleven days after the initial chill.

After the air-spaces have become completely filled with the exudation, if the patient continues to live, there follows a period during which the exudate becomes first decolorized, and then degenerated. This is the period of "gray hepatization." The lung remains solid, its color changes, first to a mottled red and gray, then to a uniform gray. The coloring matter is discharged from the red blood-cells and the exudate begins to de-

generate and soften. The lung is found passing from red to gray hepatization at any time between the second and the eighteenth day of the disease. It is found completely gray at any time from the fourth to the twenty-fifth day. About one-half of the cases die in the condition of mottled red and gray hepatization; about one-fourth in the condition of gray hepatization.

If the patients recover the exudate undergoes still further degeneration and softening and is removed by the lymphatics. This is the stage of "resolution." It should commence immediately after defervescence and be completed within a few days. But it may not begin until a number of days after defervescence, or it may be unusually protracted.

Modifications of the inflammation.—The lung, instead of being freed from the exudate at the regular time, may remain in the condition of gray hepatization for weeks.

The quantity of inflammatory products may be so great that the blood-vessels are compressed and portions of the lung become necrotic.

There may be an excessive production of pus-cells, with infiltration of the walls of the air-spaces and of the stroma of the lung.

The inflammatory product within the air-spaces, instead of consisting only of fibrine, pus, and epithelium, may consist also of organized connective tissue.

The bronchitis may be developed in an unusual degree, and involve not only the bronchi of the inflamed lung but also those of the other lung.

The pleurisy may be unduly developed at any time in the course of the pneumonia, or after it has subsided.

There may be an excessive production of fibrine, or of fibrine and serum, or of fibrine, serum, and pus.

There may be gangrene of part of the inflamed lung.

Causes.—Lobar pneumonia occurs in nearly all climates, warm as well as cold. In the United States it is said to be more common in the South than in the North.

In New York it prevails in the winter and spring, and the colder the weather the more numerous are the cases of pneumonia. In some cases there is a distinct history of exposure to cold. There are occasionally local epidemics of the disease, and rarely it seems to be contagious.

The disease seems to have the characters of an infectious inflammation which requires for its production an exciting cause of inflammation and the presence and growth of the *diplococcus pneumoniae*. It may retain throughout the characters of a local inflammation, or it may be accompanied by a general infection.

Children under five years of age usually have broncho-pneumonia ; children between the ages of five and fifteen may have either broncho-pneumonia or lobar pneumonia ; adults usually have lobar pneumonia.

Symptoms.—Physical signs. During the stage of congestion there is commencing dulness on percussion. The respiration is rude, or feeble. There may be a subcrepitant râle. If the bronchitis is excessive, there are coarse and subcrepitant râles, and sibilant and sonorous breathing.

During the stage of red hepatization there is, on percussion, more or less dulness, or flatness, or tympanitic resonance, or cracked-pot sound. The vocal fremitus is

regularly increased, but may be diminished. Bronchial voice and breathing are often, but not always, present. The normal breathing sounds are lost. There is often a crepitant râle. In the stage of gray hepatization the physical signs are the same as during red hepatization, the crepitant râle may persist, or may disappear.

During the stage of resolution the dulness diminishes, the normal respiratory murmur begins to return, there are crepitant and subcrepitant râles.

In old persons the physical signs are often imperfectly developed, and sometimes absent altogether.

Rational symptoms. In from one-sixth to one-third of the cases there are prodromic symptoms. Chilliness, moderate fever, general malaise, oppression of the chest are present and continue for from one to four days. These symptoms probably correspond to a protracted period of congestion.

In about ninety per cent. of the cases there are, during the first twenty-four hours, one or more decided chills, and it is from the chill that we usually reckon the days of the disease.

The temperature rises at once, and often reaches its maximum on the first or second day. It continues, with morning remissions and evening exacerbations, until defervescence. Sometimes the temperature is the highest on the day before defervescence. The ordinary temperature of lobar pneumonia is about 104° F.

Defervescence may take place at any time from the second to the eighteenth day. It is most common on the seventh day; next on the fifth, eighth, sixth, and ninth days in order. It usually begins in the evening,

and the temperature falls to or below the normal in from six to forty-eight hours.

The height of the temperature is usually in proportion to the severity of the disease. But some cases do well in spite of very high temperatures; some cases get worse with a falling temperature; some cases die with a temperature below the normal; there are rare cases without any rise of temperature. The fever may be prolonged by the extension of the inflammation to fresh portions of the lung.

There is often pain over the inflamed lung, referred to the region below the nipple, and developed within twelve hours after the chill. There is much difference in the patients as to the severity of the pain. In some cases there is no pain until the patients begin to cough.

The character of the heart's action and of the pulse is of great importance. The pulses should be about 100, and full. It may be too rapid, or too slow, or too full. On the day before defervescence there is danger of failure of the heart's action, with venous congestion, cerebral symptoms, or sudden death.

The breathing, at the invasion of the disease, is oppressed and rapid; later, rapid, more or less labored, and insufficient. The badness of the breathing is in proportion to the extent of lung involved, or to the severity of the complicating bronchitis.

The cough is developed early in the disease, or later. It is often most troublesome as resolution begins. In some cases the cough is trifling, or absent.

The characteristic sputa are little rounded, viscid pellets of red, yellow, or brownish color, mixed with thin

fluid mucus. In bad cases there is expectoration of a diffuent, dark-colored fluid. The sputa may be nothing but ordinary mucus, or there may be no sputa. In rare cases the patients cough up pure blood at the beginning of the disease.

The cheeks are usually flushed. The tongue is coated, sometimes dry. There is loss of appetite, nausea, sometimes vomiting.

The urine is diminished in quantity, high colored, sometimes contains a little albumen and a few casts.

Headache, restlessness, and sleeplessness are present in many of the cases; delirium, stupor, and coma in a smaller number.

The degree of prostration varies with the severity of the disease. There are rare cases in which patients continue to be up and about after the lung is consolidated.

Complications.—At any time in the course of the pneumonia, or after it has subsided, there may be developed pleurisy with effusion, or empyema, on the same side as the pneumonia.

If an excessive general bronchitis accompanies the pneumonia it changes the physical signs, adds to the dyspnoea, and raises the temperature.

Acute pericarditis is always a serious complication. The only evidence of it may be the greater illness of the patient; or there may be cyanosis, rapid breathing, a rapid and feeble pulse, pain over the heart, and the characteristic friction sound.

A previously existing chronic endocarditis adds much to the dangers of the pneumonia. If it is an aortic or mitral stenosis the characteristic murmur is often absent.

Acute meningitis is an infrequent complication. It may, or may not, be attended with characteristic symptoms.

Jaundice may occur, either with mild or with severe cases.

If the kidneys have been previously normal the pneumonia only produces a mild degeneration, or an exudative nephritis. But if a chronic nephritis already exists the pneumonia is apt to bring on an exacerbation of the nephritis, with serious renal symptoms.

The course of the disease.—1. The simple cases. The patients have one or more chills, a temperature of 100°–104° F, vomiting, pain in the side, prostration, breathing from 25 to 40, cough, the characteristic sputa, a pulse of 90 to 110 and full, sleeplessness, restlessness, and the characteristic physical signs. Defervescence and resolution take place at the regular times. Or, on the day before defervescence, there is heart failure and death.

2. The cases run the regular course, but the physical signs are not developed until after several days.

3. The inflammation, instead of remaining confined to the lobe where it began, may extend to other portions of the lung. Each extension of the inflammation is attended with an exacerbation of the symptoms.

4. The inflammation from the outset involves the larger part of both lungs, and the patients die within a few days.

5. Resolution, instead of beginning within one or two days, may be delayed for one, two, or three weeks.

6. There are cases in which the patients behave as

if poisoned by an extension of the infection from the lung, and this without reference to the extent of lung inflamed. They have marked cerebral symptoms, a dry tongue, high temperature, a rapid and feeble pulse, rapid emaciation. This form of the disease is usually fatal.

7. There are cases, otherwise normal, but with excessive delirium, which may continue even after defervescence. Sometimes the patients remain insane after recovering from the pneumonia.

8. The course of the disease is altered by the complicating lesions—meningitis, bronchitis, pleurisy, pericarditis, endocarditis, and nephritis.

9. In old persons the physical signs are obscure, the cough is slight or absent, the disease is badly borne.

Modes of death.—The patients die with heart failure just before defervescence ; from the extent of the inflammation ; from general poisoning ; from one of the complications ; from thrombosis of the coronary arteries.

Duration.—In the cases which recover, defervescence takes place at any time from the second to the eighteenth day, most frequently on the seventh or fifth. Resolution is accomplished within a few days after this, but may be delayed up to three weeks.

In the fatal cases death may take place at any time from five hours to thirty days—most frequently on the seventh, eighth, and tenth days.

In persons over seventy death is most common on the fifth, sixth, and seventh days.

Treatment—Attempts to check the pneumonia at its beginning are occasionally made and are sometimes suc-

cessful. For this purpose venesection and large doses of calomel are employed.

For the most part, however, we are content to treat the symptoms. In a considerable number of cases, which run a regular course, nothing is necessary but careful nursing.

Bad breathing may be relieved by cupping the entire chest, or by giving small doses of calomel and opium. Excessive restlessness, sleeplessness, and pain can be mitigated by opium, the bromides, or assafoetida.

The temperature is best left alone, even if it is high.

An excessively full pulse can be relieved by small doses of aconite or veratrum viride.

Failure of the heart's action requires the use of alcoholic stimulants, or of combinations of digitalis, convallaria, and the iodide of potash.

II. SECONDARY LOBAR PNEUMONIA.

Lesions.—The inflammation does not involve an entire lobe, but portions of one or more lobes, so that we find scattered areas of hepatization with the rest of the lung congested. The air-spaces and small bronchi are filled with fibrine, pus, and epithelium.

Causes.—This form of pneumonia is common with injuries and inflammation of the brain and spinal cord, after surgical operations, with any disease or injury which confines a patient to bed, and with the infectious diseases.

Symptoms.—In some cases the pneumonia gives neither rational symptoms nor physical signs. In others there are chills, fever, rapid breathing, pain, cough, and expectoration, and the physical signs of consolidation of the lung.

III. THE PNEUMONIA OF HEART DISEASE.

This is a chronic inflammation of both lungs, produced by valvular lesions of the heart, by dilatations of the ventricles, and by aneurisms of the arch of the aorta. It is most common with mitral stenosis.

Lesions.—The lungs are small, there are often old pleuritic adhesions, there may be serum in the pleural cavities. The texture of the lungs is leathery and dense; they are dry and not congested; they are of a pink or salmon color, mottled with brown or black. They may be partly consolidated. There may be one or more hemorrhagic infarctions. The capillaries are large and tortuous, the walls of the air-spaces are thickened, the air-spaces are partly or completely filled with epithelial cells.

Symptoms.—The patients have dyspnœa, cough, mucous and bloody sputa, sometimes continued expectoration of large quantities of pure blood. The only physical signs are dulness on percussion and imperfect breathing, with sometimes a few râles.

IV. BRONCHO-PNEUMONIA.

Synonymes: Capillary Bronchitis; Lobular Pneumonia; Catarrhal Pneumonia.

Lesions.—The essential and constant lesion of broncho-pneumonia is an inflammation of the walls (not the mucous membrane) of the bronchi, and of the air-spaces immediately surrounding the inflamed bronchi. The walls of the bronchi are thickened and infiltrated by a growth of new cells. The walls of the air-spaces are thickened, their cavities are filled with fibrine, pus, and

epithelium, or with new connective tissue. The inflammation involves the medium-sized and smaller bronchi of both lungs, but is not everywhere equally severe; in some parts of the lungs the lesions are much more marked than in others. In some of the cases there are no other changes, except some general congestion of the lungs. In other cases there may be added: A catarrhal inflammation of the mucous membrane of the bronchi, diffuse consolidation of parts of the lung, pleurisy, dilatation of the inflamed bronchi, areas of atelectasis, simple or tubercular inflammation of the bronchial glands.

The character of the inflammation is such that resolution is slow, and the acute inflammation may be followed by a chronic one.

If the broncho-pneumonia becomes chronic the walls of the bronchi remain thickened, the bronchi remain dilated, the peribronchitic zones of pneumonia persist. If a larger part of the lung has been consolidated, this consolidation persists, with a diffuse growth of connective tissue, and sometimes with thickening of the pulmonary pleura.

Causes.—Broncho-pneumonia is the ordinary pneumonia of children; it is frequent in young persons and occasionally seen in adults.

It occurs as a primary inflammation, is often secondary to measles and whooping-cough, less frequently to the other infectious diseases. It is most frequent during the cold and wet months of the year; in some cases there is a history of exposure to cold, in others no exciting cause is discoverable.

The same patient may suffer from several attacks of the disease.

Symptoms.—In very young infants the only symptoms are fever, prostration, and rapid breathing. The disease is almost certainly fatal.

In older children there may be first the measles or whooping-cough, or a coryza, or pharyngitis, or catarrhal bronchitis, and then the broncho-pneumonia. Or the disease may begin without any preceding morbid conditions. In some of the cases the invasion is marked by general convulsions. There is a rapid rise of temperature, the patients are sleepless, restless, or delirious, the pulse is rapid, the breathing is rapid and insufficient, the face is flushed, the tongue is coated and sometimes dry, there may be vomiting, sometimes there is pain over the chest. There may be no cough, or cough with mucous sputa. The urine may contain a little albumin and a few casts. The disease varies in its severity, but most of the patients are seriously ill.

The physical signs vary with the condition of the lung. If the inflammation is limited to the walls of the bronchi and to the air-spaces immediately surrounding them, there are no physical signs. If a catarrhal bronchitis is added, there are coarse and subcrepitant râles. If there is a diffuse pneumonia, with consolidation of a considerable part of the lung, there are dulness on percussion and bronchial voice and breathing; if there is fibrine on the pleura, there are crepitant and subcrepitant râles. The signs of consolidation are usually developed between the second and fifth days. The constitutional symptoms continue for from seven to fourteen days, and then gradually or quickly subside. Resolution follows, and is accomplished in from seven to fourteen days more.

This is the course of the disease in the regular cases which recover.

The bad cases die at any time from the second to the fourteenth day.

In the cases which recover, resolution is sometimes delayed for several weeks, and the children then continue to be pale, irritable, prostrated, without appetite, sometimes with a little afternoon fever.

In some cases the inflammation extends and involves successively different portions of the lungs.

The cerebral cases.—In many of the cases of broncho-pneumonia there are cerebral symptoms—convulsions, restlessness, and delirium—but in some patients these symptoms are developed to such a degree, and are so out of proportion to the pulmonary symptoms, that the cases require a separate description.

The symptoms resemble those of an acute or a tubercular meningitis. They may begin and go on acutely with high fever, prostration, one or more convulsions, alternating delirium and stupor. Or the course is more subacute, loss of appetite, vomiting, moderate prostration, not very high fever, alternating stupor and delirium. These symptoms may continue for from two to ten days before there are any pulmonary symptoms. Then, as the pulmonary symptoms are developed, the cerebral symptoms subside.

If, after the subsidence of the acute broncho-pneumonia a chronic inflammation persists, the children begin to improve, but yet do not get well.

In some the cough and the physical signs continue, the appetite is poor, the children do not gain flesh and

strength, but yet they are not sick in bed—often not confined to the house.

In others the same symptoms exist, there is also an irregular fever, and the patients are sick in bed.

Of these protracted cases some recover entirely ; some recover with permanent consolidation of a portion of the lungs ; some die exhausted by the disease ; some go on to have a chronic interstitial pneumonia which lasts for many years.

In some cases of acute broncho-pneumonia, the accompanying inflammation of the bronchial glands may be of tubercular character, and this may later serve as the starting-point of a general tuberculosis.

Treatment.—If the inflammation is confined to the walls of the bronchi and the air-spaces around them, counter-irritation of the wall of the chest and antiphlogistic remedies are not likely to be of service. If, on the other hand, catarrhal bronchitis and general congestion of the lungs are present, with rapid and labored breathing, it may be well to use cups, or irritating liniments, or poultices over the chest, and to give repeated small doses of calomel or the sulphate of magnesia. Throughout the disease good results may be obtained from the continued use of ipecac in small doses.

The sleeplessness, restlessness, and delirium are best relieved by opium ; in children who cannot take opium, we may use the bromides or assafoetida. The disposition to convulsions seems to be lessened by the use of the bromides. If the pulse is too full, with high temperatures, the children are made more comfortable by the tincture of aconite. Alcoholic stimulants are not to be

used unless there is decided failure of the heart's action. The feeding and nursing of the child are of the greatest importance.

If resolution is delayed, or if the broncho-pneumonia persists, we employ iron, quinine, mineral acids, oxygen, cod-liver oil, and, above all, change of air.

In adolescence the clinical picture of broncho-pneumonia is the same as it is in children, but the cerebral symptoms are not developed to the same extent, and they are more likely to cough up blood.

In adults the disease presents itself to us under several different forms :

1. The patient has an ordinary attack of catarrhal bronchitis lasting for several days. Instead of getting well promptly, however, the patients continue to cough and to feel sick, and, on examining the chest, we find a circumscribed area where there is dulness on percussion and loud, high-pitched voice. This consolidation of the lung does not, however, last very long, and the patients make a good recovery.

2. The patients are suddenly attacked with a very severe and general broncho-pneumonia. There are chills, a rapid rise of temperature, headache, pains in the back and chest, vomiting, great prostration, a rapid pulse which soon becomes feeble, very bad breathing—rapid, labored, and insufficient—venous congestion of the skin and of the viscera, cough at first dry, then with profuse mucous and blood-stained sputa, sleeplessness, restlessness and delirium, and albumin in the urine. There are coarse subcrepitant and crepitant râles over both lungs, sibilant and sonorous breathing ; the percussion-note is

normal, or exaggerated, or dull. The disease lasts for from seven to fourteen days; it is very apt to prove fatal.

Treatment.—The most efficient remedies are the energetic use of wet or dry cups over the entire chest, the administration of calomel or the sulphate of magnesia in small and repeated doses, ipecac, the inhalation of oxygen-gas, and stimulants.

3. There is a form of broncho-pneumonia in adults which resembles lobar pneumonia. There is a general catarrhal bronchitis, with broncho-pneumonia and consolidation of one or more lobes. The symptoms and physical signs are like those of lobar pneumonia, but with some difference. The invasion of the disease is not as sudden, the pulse is more rapid, the cerebral symptoms are more constant, the expectoration is like that of bronchitis, the physical signs are more slowly developed, the duration of the disease is rather longer and resolution is slower.

4. There is a form of broncho-pneumonia which resembles acute phthisis.

The patients have a cough with expectoration, at first mucous, afterward muco-purulent. There is a moderate fever, with evening exacerbations and sweating at night. The patients steadily lose flesh and strength. The physical signs are those of bronchitis and of consolidation of parts of the lung. The disease is protracted, continuing as long as ten weeks, and is apt to prove fatal.

5. Broncho-pneumonia, especially of the lower lobes, is secondary to the infectious diseases, to injuries, to surgical operations, and to any conditions which confine patients to bed.

THE LUNGS.

INTERSTITIAL PNEUMONIA.

MODERATE degrees of inflammation of the lung, with the production of interstitial connective tissue, are common. Such a development of interstitial pneumonia as to constitute a disease by itself is comparatively rare.

Causes.—Well-marked interstitial pneumonia follows attacks of broncho-pneumonia ; severe attacks of pleurisy which leave behind extensive adhesions ; chronic bronchitis ; and the inhalation of the dust of coal or stone.

Lesions.—The inflammatory changes are regularly confined to one lung, except in the cases of inhalation pneumonia. This lung is thickly covered with old adhesions, its pulmonary pleura is thickened, the lung itself is small. Bands of dense fibrous tissue extend from the pleura into the lung. There is a growth of connective tissue in the septa between the lobules, around the bronchi and the blood-vessels, and in the walls of the air-spaces. The cavities of the air-spaces are compressed and obliterated. The mucous membrane of the bronchi is the seat of chronic catarrhal inflammation. The walls of the bronchi may be thickened and their lumen narrowed ; or their walls may be thinned with the formation of bronchi-ectasiæ.

The other lung is large and emphysematous.

Symptoms.—The patients have a cough with mucous expectoration. The cough becomes more constant and troublesome as the disease progresses. The expectoration becomes muco-purulent, sometimes fetid. If the bronchi become dilated, the muco-pus accumulates in them and is coughed up at intervals in large quantities. There may be occasional hæmoptyses. There is dyspnœa on exertion, at first slight, later more marked. There may be uncomfortable feelings or actual pain over the affected side of the chest. There is gradual loss of flesh and of strength. Neither laryngitis nor diarrhœa belong to the disease. There is no fever except with exacerbations of the bronchitis.

The affected side of the chest is retracted, the other side is enlarged, the heart is displaced, the vertebral column is curved laterally. There is, on percussion, dulness, or flatness, or tympanitic resonance. The vocal fremitus is increased. The breathing is sonorous,⁴ sibilant, bronchial, cavernous, or amphoric, according to the condition of the bronchi and the degree of consolidation of the lung. There are pleuritic creaking sounds, subcrepitant, coarse, or gurgling râles.

The disease is one which lasts for many years, and the patients usually die with some acute inflammation of the previously healthy lung.

Treatment.—The most efficient treatment is that the patient should reside permanently in a climate where he is able to live out of doors, and where his bronchitis does well.

If this cannot be done, we attend to the bronchitis and the nutrition of the patient as well as we can.

SYPHILITIC PNEUMONIA.

Constitutional syphilis may give rise to inflammation of the lung.

The most ordinary form of syphilitic pneumonia is that of a chronic interstitial inflammation with the production of new connective tissue. The distribution of the inflammation varies :

1. There is an interstitial pneumonia beginning around the large bronchi and blood-vessels at the root of the lungs, extending to the interlobular septa and the walls of the air-spaces, and resulting in the conversion of the central portions of one or both lungs into dense fibrous tissue.

2. The inflammation follows the type of a broncho-pneumonia, with thickening of the walls of the bronchi and small zones of peri-bronchitic pneumonia.

3. With more or less interstitial pneumonia, there is a syphilitic inflammation of the walls of the trachea and large bronchi. These walls are thickened in some places, ulcerated in others, so that either the condition of stenosis or dilatation may be established.

4. With the interstitial pneumonia there may be associated the production of gummy tumors, or an obliterating endarteritis with areas of coagulation necrosis.

We also occasionally meet with pneumonias of the type of lobar pneumonia or broncho-pneumonia, which run an acute but irregular course, and which seem to be caused by syphilis.

TUBERCULOSIS.

Persons are said to suffer from tuberculosis if there are going on, in one or more parts of their bodies, tuber-

cular inflammations. To such tubercular inflammations may, or may not, be added evidences of systemic infection.

Tubercular inflammation is an inflammation accompanied with the growth of the tubercle bacillus. The inflammation itself is of the exudative, or productive type, or of both combined.

The tubercle bacilli are little beaded rods, 2 to 5 mikrom. in length. They are found in the inflamed tissues, the inflammatory products, the lymphatics, and the blood-vessels.

The exudative form of inflammation is accompanied with the production of serum, fibrin, and pus.

The productive form is accompanied with the production of round-celled tissue, of fibrous tissue, or of tubercle tissue. Tubercle tissue is composed of a basement substance forming a meshwork enclosing large polygonal cells, and of giant cells. It is arranged in the form of little spherical bodies—tubercle granula—or of diffuse infiltrations.

With tubercular inflammations there is often associated obliterating endarteritis, the new tissue produced is of low vitality, so we find that tubercular inflammations are regularly attended with necrosis and cheesy degeneration.

There is also a well-marked disposition on the part of tubercular inflammation to continue and to become chronic.

Tubercular inflammation may be diffuse, involving large areas of tissue ; or it may be circumscribed, involving a number of small areas. In the latter case the little foci composed of products of inflammation are called miliary tubercles.

Causes.—There is a well-marked predisposition in some individuals to tuberculosis. This disposition may be either hereditary or acquired.

The most direct exciting cause is inoculation with the tubercle bacilli. This is the way in which the disease is experimentally produced in animals; but in human beings it is an exceptional method of production.

In the human being there are usually required for the production of tuberculosis: a predisposition on the part of the individual, one of the ordinary causes of inflammation, and the growth of the tubercle bacillus.

The tubercular inflammation acquired in this way may remain confined to some one part of the body and retain throughout the characters of a local inflammation. It may, after a time, be followed by the development of successive tubercular inflammations in other parts of the body. It may be accompanied with the evidences of systemic infection. It may be followed by a sudden development of tubercular inflammation in many parts of the body at the same time. There may be a sudden development of tubercular inflammation in many parts of the body, at about the same time, not preceded by a local tuberculosis.

ACUTE GENERAL MILIARY TUBERCULOSIS.

Lesions.—There is a formation of miliary tubercles in many different parts of the body, at about the same time, with more or less diffuse exudative inflammation of the same parts of the body. The tubercles are found most constantly and in the largest numbers in the lungs, liver, spleen, and serous membranes.

In the lungs the bronchi are congested and coated with mucus ; the walls of the air-spaces are congested, the epithelial cells which line them are increased in size and number, some of the air-spaces are filled with epithelium, fibrin, and pus ; there may be fibrin on the pulmonary pleura.

The miliary tubercles are present in large numbers and distributed through both lungs. They are small, or large ; gray, white, or yellow ; single or aggregated. They are composed of :

1. Groups of air-vesicles filled with granular matter, pus-cells, and epithelium.
2. Groups of air-spaces of which the walls are infiltrated with tubercle tissue, and the cavities filled with tubercle tissue, or other inflammatory products.
3. Infiltrations of the walls of small bronchi with tubercle tissue, or round-celled tissue, the infiltration often extending to the walls of the adjacent air-spaces.

There are also miliary tubercles in the pulmonary pleura, in the septa between the lobules, in the walls of the bronchi, and in the walls of the veins.

Causes.—Acute general tuberculosis may be due to infection from a pre-existing local tuberculosis, or it may be a primary general infection.

Symptoms.—There are local symptoms due to the inflammations of the pia mater, the lungs, and the peritoneum ; and general symptoms due to the systemic infection. The cases vary as to the predominance of the local or the general symptoms.

The invasion of the disease may be acute, the patient at once confined to bed with a high temperature ; or

subacute, the patient able to be up, and with but little fever. The subsequent course of the disease may be acute and short, with high temperatures ; or subacute and prolonged, with lower temperatures.

The fever may follow the continued, or remittent, or intermittent types. The temperature may be high or low, or not above the normal.

The prostration may be marked from the first, or it may be days or weeks before the patient is confined to bed. The pulse and the heart action are rapid and feeble throughout the disease.

The tongue is coated, late in the disease brown and dry. The breathing is rapid.

There is loss of appetite, nausea, and sometimes vomiting. The bowels may be normal, or constipated, or loose.

There may be complicating acute degeneration of the kidneys, or exudative nephritis.

Sooner or later alternating stupor and delirium, extreme emaciation, and the typhoid state are developed.

There are often miliary tubercles in the choroid coat of the eye, which can be seen with the ophthalmoscope.

The disease continues only for a few days, or for weeks or months. It always terminates fatally.

Varieties.—In children the most common form of the disease is that which follows the type of a tubercular meningitis. Less frequently the disease is not attended with cerebral symptoms, but with rapid and insufficient breathing, with or without the physical signs of bronchitis.

In adults the disease may take the form of a tubercular meningitis.

Or there may be marked pulmonary symptoms : cough

with mucous sputa ; rapid and oppressed breathing ; pain over the chest ; a crepitant râle if there is pleurisy ; coarse and subcrepitant râles with the bronchitis ; dullness on percussion if there is consolidation. If there has been a pre-existing chronic phthisis, the physical signs of this will persist. Occasionally the symptoms are those of a tubercular peritonitis.

Rarely the tubercles are confined to the lymphatic glands, and the disease resembles pseudo-leukæmia. The patients lose flesh and strength, they become anæmic, they have an irregular fever, and the lymphatic glands throughout the body become enlarged.

Not infrequently there are no local symptoms, and the disease resembles a typhoid or malarial fever. The patients lose flesh and strength ; they have fever of continued, remittent, intermittent, or irregular type, and finally pass into the typhoid state.

In some of the cases of this type the symptoms of the tuberculosis are masked by those of some severe complicating disease.

TUBERCULAR PNEUMONIA.

Independently of general tuberculosis, the lungs may become the seat of localized tubercular inflammations. These inflammations may conveniently be described under the names of Sub-acute Miliary Tuberculosis, Chronic Miliary Tuberculosis, Acute Phthisis, and Chronic Phthisis.

I. SUB-ACUTE MILIARY TUBERCULOSIS.

Lesions.—There are miliary tubercles of the characters already described in the lungs. These tubercles

may occupy only a part of one upper lobe, or the larger part of both lungs. There may be also a general or localized catarrhal bronchitis; but usually no pleurisy or diffuse pneumonia are present.

Causes.—Subacute miliary tuberculosis may be developed without discoverable cause in persons who have the tubercular predisposition, or it may be secondary to a chronic miliary tuberculosis.

Symptoms.—These are in proportion to the extent of lung involved.

If only one upper lobe is involved, the patients have afternoon fever with night-sweats, a rapid pulse, more or less dyspnoea, cough with mucous expectoration, sometimes hæmoptyses, and gradual loss of flesh and strength. Although they are evidently seriously ill, they are often not confined to bed.

If both lungs are involved, the patients lose flesh and strength more rapidly, they develop alternating delirium and stupor, and pass into the typhoid condition.

As there is no pleurisy, nor consolidation, the physical signs are limited to the coarse and subcrepitant râles of the bronchitis, and the altered quality of the breathing due to the miliary tubercles.

If both lungs are involved, the symptoms continue and the patients die after a few weeks. If only one upper lobe is involved the disease may last for a few months and the patients then recover, or it may be succeeded by chronic miliary tuberculosis.

Treatment.—The most efficient plans of treatment are : to send the patients to a proper climate, and to cause

them to take the largest possible quantity of food, especially of fats.

II. CHRONIC MILIARY TUBERCULOSIS.

Lesions.—The morbid process begins at the apex of one lung and then slowly extends, either progressively or in attacks, until a larger part of the lungs is involved.

In the simplest form of the disease the only change in the lungs is the formation of miliary tubercles. These tubercles are harder and denser than those found with general tuberculosis, or with subacute pulmonary tuberculosis. They are composed of tubercle tissue, or round-celled tissue, or connective tissue, or are in the condition of cheesy degeneration.

Usually, however, in addition to the miliary tubercles there are other changes in the lungs. These additional lesions begin in the same part of the lung where the tubercles are formed, and accompany the development of the tubercles in fresh parts of the lungs.

There may be a localized catarrhal bronchitis.

There may be an inflammation of the walls of the bronchi with partial destruction of these walls, and the formation of cylindrical or sacculated bronchi-ectasiæ. The walls of the cavities thus formed may be converted into connective tissue, or they may remain suppurating and necrotic.

There may be an interstitial pneumonia with the production of new connective tissue, the obliteration of the air-spaces, and the consolidation of portions of the lung.

There may be dilatation of the air-spaces of the portions of the lungs which are not consolidated.

There may be thickening of the pulmonary and costal pleura, with connective-tissue adhesions.

While the morbid process begins as a localized tubercular inflammation of the lung, and often retains throughout this local character, yet it may also happen that from this local lesion other parts of the body may be infected. Tubercular laryngitis, and tubercular inflammation of the solitary and agminated glands of the small intestine, often complicate the pulmonary lesion, and sometimes even acute general tuberculosis is produced.

Symptoms.—The disease may follow a subacute tuberculosis, or it may be chronic from the outset. If it is chronic from the outset it gives at first no symptoms. Indeed, there seems to be reason to believe that a chronic miliary tuberculosis of small extent may run its entire course and get well without giving any symptoms at all. Usually, however, sooner or later, symptoms are developed.

The first symptom may be one or more small hæmoptyses, less frequently a large hæmoptysis, the patient otherwise feeling very well. Or the first symptom may be a cough which continues for weeks or months, and does not get better. Or the patient may simply lose flesh and strength, with afternoon fever, and a rapid pulse.

At this early stage of the disease there may be no physical signs. If any such signs are present they are due to thickening of the pleura, to miliary tubercles alone or combined with fibrous tissue, and to localized bronchitis, all confined to the apex of the lung. We may find, therefore, at one apex : retraction of the chest above and below the clavicle, dulness on percussion, subcrepitant

râles, a high-pitched and louder voice, the breathing diminished, or of altered quality, or with prolonged expiration.

When the disease is established it may run a mild course. The inflammation remains confined to one apex. The patients have cough, with or without mucous sputa; loss of appetite, flesh, and strength; pain over the inflamed lung; an irregular fever; a rapid pulse; hæmoptyses; the physical signs due to the pleurisy, the localized bronchitis, the miliary tubercles, and the interstitial pneumonia. In some of the cases the symptoms are not at any time severe; in others the symptoms are so out of proportion to the lesion as to indicate a systemic infection. The symptoms continue for months, or for one or two years. Then they subside, the patients recover, but the inflamed portion of the lung is permanently changed, and they are liable to fresh attacks of the disease.

Or the disease may run a severe course. The inflammation extends and involves more and more of the lungs. The pleuritic adhesions are more extensive, the bronchitis, the interstitial pneumonia, and the tubercles involve more of the lungs, cavities are formed by the dilatation of the bronchi, and the walls of these cavities may suppurate.

The patients get worse from year to year, but with intervals of improvement. The physical signs correspond to the extent of the pleurisy, the bronchitis, the consolidation, and the cavities.

The hæmoptyses may be repeated, either from the bronchi, or from eroded vessels in the walls of bronchi.

The cough may depend upon the pleurisy, the bronchitis, or the cavities. There is no expectoration, or the sputa are mucous, or muco-purulent. They usually contain tubercle bacilli.

There is dyspnoea according to the extent of lung inflamed and the complicating emphysema.

An irregular fever, higher in the afternoon and with sweating at night, comes and goes throughout the disease. The pulse is habitually rapid. There is more or less pain over the inflamed lung. In women menstruation is apt to cease altogether. There is loss of appetite, sometimes vomiting and marked emaciation.

There may be evidences of tubercular infection from the lungs, or of pyæmic infection from the suppurating walls of the cavities.

Complicating laryngitis, or enteritis, may add their symptoms.

The disease, when once established, goes on continuously, or with intermissions for years.

The best marked cases are those with cavities and considerable consolidation of the lung. The obscure cases are those in which the miliary tubercles constitute the only lesion, or in which the cavities are small.

There are cases in which the laryngitis, with its characteristic symptoms, is the marked feature of the disease, while the pulmonary lesions and symptoms are but little developed.

There are rare cases in which, in the same way, the intestinal lesion is disproportionately developed.

The Prognosis in the early periods of the disease, with little evidence of systemic infection, is good. As more

of the lungs is involved, as cavities are formed, as the complicating laryngitis and enteritis are developed, and the evidences of systemic infection become manifest, the prognosis is worse and worse.

Treatment.—The curative treatment of chronic miliary tuberculosis is embraced in two principal things—climate and feeding.

The selection of climate is to be made with reference to the individual, and not with reference to his lungs. It should be a climate where he feels well, eats well, sleeps well, and gains flesh and strength. If no one climate answers this purpose, the patient should travel from place to place. The climatic treatment should be continued, if possible, for two full years.

The feeding consists in enabling the patient to eat and digest considerable quantities of wholesome food and of fats. To do this, the most minute attention is necessary to the functions of the stomach, the intestines, and the liver. Great care should be taken to avoid all medicines which interfere with the patient's ability to eat and digest food.

It may also be necessary to alleviate symptoms.

The cough may be due to pharyngitis, laryngitis, pleurisy, bronchitis, or cavities.

The pharyngitis and laryngitis are to be treated by local applications; the pleurisy by counter-irritation over the chest; the bronchitis and cavities by inhalations of compressed air, the mineral acids, the preparations of turpentine and strychnia. It may be necessary to use opium, but this should be avoided as long as possible.

If the patients are anæmic, they require the administration of iron and the inhalation of oxygen.

The smaller hæmoptyses require no treatment. The larger ones may be restricted by the use of ergot, ipecac, or gallic acid.

The fever and night-sweats may be made less severe by the use of antifebrin alone, or combined with quinine, or arsenic ; by the mineral acids, belladonna, and the oxide of zinc ; by sponging off the body with hot water at night.

For the diarrhœa we employ a restricted diet, the bichloride of mercury, ipecac, iron, and arsenic, opium by itself, or combined with castor-oil, bismuth, the acetate of lead, or camphor.

ACUTE PULMONARY PHTHISIS.

Acute consumption. Acute catarrhal phthisis. This name is used to designate an acute tubercular inflammation of the lungs combined with other non-tubercular inflammatory changes.

Lesions.—1. In one or more lobes there may be miliary tubercles and a diffuse consolidation, due to the filling of the air-spaces with fibrin, pus, and epithelium.

2. There is a tubercular broncho-pneumonia involving the smaller bronchi of one or more lobes, and zones of air-spaces surrounding these bronchi. The walls of the bronchi and of the surrounding air-vesicles are infiltrated with tubercle tissue.

3. Besides the tubercular inflammation of the walls of the bronchi and of the air-spaces surrounding them, there are a general catarrhal bronchitis, diffuse consolidation of the lung by the filling of the air-spaces with fibrin, pus, and epithelium, and fibrin on the pulmonary pleura.

4. Besides the tubercular broncho-pneumonia, the diffuse consolidation, and the pleurisy, there are small or large portions of dead lung-tissue in the condition of coagulation necrosis. These areas pass into the condition of cheesy degeneration, and are surrounded by zones of tubercle tissue, or of round-celled tissue ; or they soften and form cavities which are in communication with the bronchi.

5. In addition to the lesions just mentioned, the walls of the bronchi are so changed by the tubercular inflammation that cylindrical or sacculated bronchi-ectasiæ are formed.

Causes.—An attack of acute phthisis may follow some previous tubercular inflammation of the lung, or it may be a primary disease. A person who has the tubercular predisposition, when exposed to the ordinary causes of inflammation of the lung, instead of having a lobar or broncho-pneumonia, has a tubercular pneumonia.

Symptoms.—The invasion may be acute or subacute.

1. *The Acute Cases.*—The patients are suddenly attacked with chills, fever, pain in the side, cough with mucous expectoration, and marked prostration. The whole appearance of the patient is that of a person attacked with lobar or broncho-pneumonia. In addition to the other symptoms there is often a profuse hæmoptysis continued for several days. Within one or two days the physical signs of the bronchitis, the pleurisy, and the consolidation of the lung are developed : Coarse, subcrepitant and crepitant râles, dulness on percussion, increased vocal fremitus, bronchial voice and breathing.

All these symptoms are continued for one or two weeks. Then there is a partial subsidence of the symptoms. After this the patients may :

(a) Continue to get worse. The fever persists, the cough is worse and accompanied with muco-purulent expectoration, there is rapid loss of flesh and strength, the patients pass into the typhoid state, with alternating delirium and stupor. The physical signs of the consolidation of the lung continue, and there are added those of the formation of cavities. The patients die at the end of a few weeks or months.

(b) The improvement is greater. The appetite returns, the patients have less fever, gain strength, are able to get out of bed, and then go on to the condition of chronic phthisis.

(c) If the inflammation is limited to the lower lobe, the quantity of the tubercular inflammation small, with no areas of coagulation necrosis, or bronchi-ectasiæ, then the inflammatory products may be absorbed, the lung return nearly to its natural condition, and the patients recover.

2. *The Subacute Cases.*—The patients have cough with mucous expectoration, dyspnœa, afternoon fever, loss of appetite, flesh, and strength, often large hæmoptyses. They are not sick enough to remain in bed all the time. The physical signs are those of consolidation of part of one of the upper lobes, or of localized or general bronchitis without consolidation.

If there is consolidation of part of one of the upper lobes, after a time one or more cavities are formed. The patients then go on to have chronic phthisis ; or they may

have successive attacks of acute phthisis involving fresh portions of the lung.

If there is only a tubercular broncho-pneumonia without consolidation, this may continue for one, two, or three years, and then subside, and the patients get permanently well. Or the broncho-pneumonia may continue, bronchi-ectasiæ are formed, the lung between the inflamed bronchi becomes the seat of chronic inflammation, and the patients go on to have chronic phthisis.

The Prognosis of acute phthisis is unfavorable. The only cases which recover are those in which there is tubercular broncho-pneumonia without diffuse consolidation of the lung ; and those in which there is consolidation of one lower lobe with but little tubercular inflammation.

Treatment.—While the inflammation of the lungs is active the patients are to be kept in bed, on a fluid diet, the hæmoptyses controlled by ergot or ipecac, the patients made more comfortable by opium or bromides.

As the acuteness of the inflammation subsides, the patients return to solid food, they get out of bed, and the question of a suitable climate for them has to be determined. In selecting a proper climate, we are guided by the condition of the lungs and the character of the patient.

In some cases it is evident that the lesions of the lung are so extensive and profound that no real improvement can be expected ; these patients are best kept at home.

In some cases the amount of lung involved is comparatively small, and although no permanent recovery can be expected, yet life may be prolonged. The proper climate for these patients is a dry, inland one, where they

are not likely to have fresh attacks of inflammation of the lung. Whether this climate should be a warm or a cold one, must be determined by the character of the individual. In the cases of tubercular broncho-pneumonia without consolidation, a cold, dry, inland climate is usually the best.

CHRONIC PHTHISIS.

Lesions.—The changes in the lungs are of the same nature as those found in chronic phthisis, but modified by the long duration of the inflammation. The pleura is thickened, the lung is consolidated, the bronchi are inflamed, there are cavities with suppurating walls formed by the dilatation of the bronchi, or the softening of areas of coagulation necrosis. Softening of portions of dead lung close to the pleura may produce perforation, escape of air into the pleural cavity, and inflammation of the pleura. There may be secondary tubercular inflammation of the larynx, and of the lymphatic glands in the wall of the large intestine.

The inflammation, although chronic, is more active at some times than at others, and there may at any time be fresh attacks of acute inflammation.

Causes.—Chronic phthisis regularly succeeds acute or subacute phthisis.

Symptoms.—The physical signs are those due to the pleurisy, the consolidation of the lung, the bronchitis, the cavities, and the perforation of the pulmonary pleura. Hæmoptyses from the bronchi are apt to mark the invasion of fresh portions of the lung; hæmorrhage from eroded vessels in the walls of cavities may occur at any time.

The cough depends principally upon the bronchitis and the suppuration of the walls of cavities. The sputa are muco-purulent and abundant.

Dyspnoea on exertion increases with the extent of lung rendered unavailable for breathing.

The fever and sweating at night seem to depend on the bronchitis and the suppuration of the cavities, rather than on the pneumonia. The functions of the stomach are often disturbed, and there may be catarrhal gastritis.

Toward the close of the disease an uncontrollable diarrhoea often sets in, either with or without ulceration of the intestine. In women menstruation is apt to cease during the continuance of the phthisis.

The laryngitis gives its characteristic symptoms.

The disease goes on nearly continuously, or with intervals of improvement.

Treatment.—The most efficient treatment is the permanent residence of the patient in a dry, inland climate, where he can lead an out-of-door life. All other treatment merely palliates the symptoms.

PULMONARY EMPHYSEMA.

I. *Interlobular, or Interstitial, Emphysema.*—This name is given to the condition of the lung produced by the escape of air into the interstitial connective tissue. This condition is most frequently a complication of broncho-pneumonia. It does not cause any recognizable clinical symptoms.

II. *Vesicular Emphysema.*—This name is given to certain chronic changes in the lungs, of which dilatation of the air-spaces forms a part.

1. *Compensating Emphysema*.—If one lung is so changed by disease that it can only partially perform its functions, the other lung becomes increased in size, and its air-spaces are dilated. This change in the lung is a healthy, rather than a morbid, one, and gives no symptoms of disease.

2. *Senile and Substantive Emphysema*.—This change in the lungs occurs as a senile change, or as a chronic inflammation; in the same way that we see similar changes developed in the endocardium, the arteries, and the kidneys.

As a chronic inflammation it begins usually in adults, in gouty persons, and in those who are disposed to chronic inflammation of the heart, arteries, and kidneys.

Lesions.—There is a chronic interstitial inflammation of the lungs, with thickening of the walls of some air-spaces, thinning of the walls of others; an increase of the epithelial cells on the walls of the vesicles; a dilatation of the air-spaces, and the formation of holes in the walls of some of the air-spaces. The dilatation of the air-spaces which has given the name to the disease does not seem to be the important part of the lesion.

The changes in the lung are sometimes attended with an obstruction to the passage of the blood through them, probably due to contraction of the branches of the pulmonary artery, or to chronic endarteritis. This obstruction to the passage of the blood through the lungs gives rise to venous congestion of the skin, stomach, small intestines, liver, and kidneys, and to dropsical effusions. After a time the right ventricle of the heart may become dilated and hypertrophied.

Acute or chronic bronchitis, pleurisy with adhesions, endocarditis, endarteritis, and chronic nephritis are often associated with emphysema.

Symptoms.—Physical Signs.

The thorax may remain of its natural shape, or there is a prominence of the costal cartilages, or the thorax assumes the barrel shape.

The percussion resonance is unchanged ; or of shorter duration, higher pitch, and altered quality ; or exaggerated, with pulmonary, or vesiculo-tympanitic quality.

The respiratory murmur is feeble, or the expiration is prolonged, or both inspiration and expiration are exaggerated. The bronchitis gives coarse and subcrepital râles, and sibilant and sonorous breathing.

The asthmatic attacks give sibilant and sonorous breathing.

The pleuritic adhesions give dulness on percussion.

Rational Symptoms. There is often difficulty in breathing, either on exertion, or constant, or spasmodic. The dyspnoea on exertion may be moderate, or so severe as to interfere seriously with the activity of the patient. The constant dyspnoea belongs to the advanced forms of the disease. It is most distressing, and is accompanied with exaggerated action and hypertrophy of the muscles of the thorax. The spasmodic dyspnoea takes the form of asthmatic attacks, or of a dyspnoea like that of Bright's disease.

Bronchitis is often a prominent feature of the disease, —either repeated attacks of acute bronchitis, or protracted chronic bronchitis.

In the advanced cases there is established a difficulty

in the passage of the blood through the lungs, which in turn is followed by venous congestion of many portions of the body, and eventually by dilatation and hypertrophy of the right ventricle of the heart. The skin is dusky-colored or livid, the ends of the fingers are swollen, there is dropsy; the liver, the spleen, the stomach and small intestines, and the kidneys are the seat of chronic congestion. The congestion of the viscera is followed by a loss of nutrition. The patients lose flesh and strength, and become anæmic.

There is a liability to short or long attacks of contraction of the smaller arteries, with increased arterial tension, headache, sleeplessness, stupor, delirium, muscular twitchings, irregular heart action, and dyspnoea.

Course of the Disease.—1. In many patients emphysema gives no symptoms at all, or only a very moderate dyspnoea on exertion.

2. In many patients the emphysema does give symptoms, but in a variety of ways.

(a) The attacks of acute bronchitis, or the chronic bronchitis, may always be the prominent symptoms.

(b) The attacks of spasmodic asthma may constitute the principal feature of the disease.

(c) The difficulty in the passage of the blood through the lungs, with the attendant dyspnoea, venous congestion, dropsy, and loss of nutrition, may render the patient very seriously ill.

(d) The complicating endocarditis, or endarteritis, or nephritis, may give their additional symptoms.

(e) There are rare and fatal cases without any pulmonary symptoms. Loss of flesh and strength, anæmia,

contraction of the arteries, with increased arterial tension and its accompanying symptoms, are developed and continue up to the time of the patients' death.

Treatment.—The chronic inflammation of the lungs is favorably affected by an out-of-door life in a suitable climate; by abstinence from alcohol, tobacco, sugars, and starches; by the use of fats; and by methodical inhalations of compressed air.

The constant dyspnoea is due to the changes in the lung, and is then to be treated by the same means which are used for the chronic inflammation; or it is due to the complicating contraction of the arteries, and is then to be treated by the drugs which dilate the arteries.

The asthmatic attacks are due to :

(a) Spasmodic contraction of the muscular coat of the bronchi. This can be relieved by inhalation of the fumes of stramonium, nitrate of potash, chloroform, and ether; by the administration of emetics, belladonna, compound spirits of ether, and opium.

(b) Congestion of the walls of the bronchi. This can be relieved by the drugs which increase the production of mucus, such as lobelia and grindelia robusta; by drugs which stimulate the heart, such as caffein, convallaria, and digitalis; and by the application of dry cups to the wall of the chest.

(c) Contraction of the smaller arteries. This can be relieved by the drugs which dilate the arteries, such as nitrite of amyl, nitro-glycerine, chloral hydrate, iodide of potash, and opium.

(d) To inflammation of the nasal passages. This is to be treated by local applications.

THE HEART.

IN examining any case of suspected disease of the heart we follow a certain routine in the physical examination. We determine the size, the impulse, the rhythm, the character of the heart-sounds and the murmurs.

The size of the heart is made out by simple or auscultatory percussion. We map out the upper, the left, and the right edges and the position of the apex-beat. Increase in the size of the heart is readily made out; diminution in its size is much more difficult to determine.

Of the impulse we notice the strength, the distinctness, the diffuseness, and the force in relation to the pulse.

Of the rhythm we notice the regularity and the rapidity.

Of the heart-sounds we notice the loudness, the distinctness, and the quality.

Murmurs are pericardial or endocardial. Pericardial murmurs are produced by the rubbing together of opposed surfaces of pericardium coated with fibrine. They are heard with the first sound, or with the second sound, or during the second silence; the murmur may be single or double. Although the murmur accompanies the heart-sounds it is not always exactly synchronous with them. There is no fixed point of maximum intensity. The quality of the murmur is superficial, rubbing or

grazing; or it is deep, soft and blowing. The murmur is not always present, but comes and goes.

Endocardial murmurs are produced by :

(a) Changes in the valves by which the valves are rendered insufficient, or stenosed, or roughened.

(b) Ventricular lesions. Inflammation of the endocardium or of the chordæ tendineæ, abnormal tendinous cords extending across the ventricle, or thrombi in the ventricles. Ventricular murmurs are systolic; their point of maximum intensity is at the apex; they are not transmitted.

(c) Unknown causes not involving any anatomical change in the heart. These murmurs are systolic, their point of maximum intensity is at the second left intercostal space or at the apex. They can be heard, in some persons, at all times; in others, only after muscular exertion; in others, only when they are anæmic. They do not indicate any disease of the heart.

PERICARDITIS.

Lesions. — The pericardium is a connective-tissue membrane like the pleura. It may be inflamed with :

The production of fibrine.

The production of fibrine and much serum.

The production of fibrine, serum, and pus.

The production of tubercle tissue.

After a pericarditis has run its course the inflammatory products may be completely absorbed; or connective-tissue adhesions are left, of small or large extent; or the inflammation becomes chronic, the purulent serum remains, and the pericardium is split up by a cell-growth; or calcific plates may be formed in the thickened pericardium.

The wall of the heart may remain unchanged, or the muscular fibres may undergo fatty degeneration, or there may be an interstitial myocarditis.

The inflammation seems usually to begin over the base of the heart.

The serum accumulates in the lower and posterior portions of the pericardial sac. As the sac is distended by the fluid the heart is pushed upward and forward, the lungs are pushed to one side, and the diaphragm is pushed down. The size of the sac may be so great that the trachea, the left bronchus, the œsophagus, or the aorta is compressed by it.

Causes.—Pericarditis is very rarely a primary inflammation. It is seen most frequently with rheumatism, pneumonia, pleurisy, and Bright's disease; less often with the infectious diseases and from injuries.

Symptoms.—The physical signs. With fibrine alone, the only physical sign is the pericardial murmur.

With distention of the pericardial sac by serum the area of precordial dulness is enlarged in all directions. The anterior wall of the chest may be bulged out. The apex-beat is carried upward. The physical signs due to the pericarditis may be complicated by murmurs due to a pre-existing endocarditis.

The rational symptoms of pericarditis are pain, rapid, feeble, and irregular heart-action, rapid and oppressed breathing, venous congestion of the skin and viscera, dysphagia, a febrile movement, and prostration.

The cases with exudation of fibrine alone give the fewest and least severe symptoms; those with large

exudations of serum give very marked symptoms ; those with serum, fibrine, and pus are the worst of all.

The prognosis in the milder cases depends more upon the primary disease than upon the pericarditis. With the large serous and purulent effusions, however, the pericarditis becomes in itself a dangerous lesion.

Treatment.—There is first the treatment of the primary disease to which the pericarditis is secondary. For the pericarditis we employ counter-irritation over the precordial region, cardiac stimulants, calomel or sulphate of magnesia as they are used for exudative inflammations. To remove excessive quantities of fluid in the pericardial sac we use cathartics, diuretics, or draw off the fluid with the aspirator.

ENDOCARDITIS.

The endocardium is a connective-tissue membrane which lines the cavities of the heart and forms the larger part of the valves. It is a thin, dense, and elastic membrane, containing but few blood-vessels. It becomes the seat of productive rather than of exudative inflammation. During foetal life the endocardium of the right heart is inflamed ; after birth, usually the endocardium of the left heart. The valves are the portion of the endocardium most frequently affected, but that which lines the auricles and ventricles and covers the papillary muscles may be inflamed independently.

We distinguish :

Acute endocarditis.

Malignant endocarditis.

Chronic endocarditis.

ACUTE ENDOCARDITIS.

Lesions.—1. The valves are swollen and succulent; the basement-substance and cells are swollen; the surface of the valves remains smooth. As the valves are swollen they are also shortened and insufficient.

2. The valves are not only swollen but there is an extensive growth of their connective-tissue cells, resulting in the formation of ulcers and of vegetations. On the roughened surfaces of the valves fibrine from the blood coagulates in considerable masses. Portions of the vegetations may become detached, pass into the aorta, and become lodged in the smaller arteries as emboli.

Causes.—Acute endocarditis is usually a complication of rheumatism, or of scarlatina.

Symptoms.—The physical signs are due to the insufficiency and roughening of the valves. So we have a mitral systolic, or aortic diastolic, or double aortic murmur.

The rational symptoms are :

Fever, prostration, disturbed action of the heart with consequent changes in the circulation, and symptoms due to embolism. The disturbed heart-action follows one of two types :

1. The heart's action is violent, tumultuous, and irregular; there is very marked venous congestion of different parts of the body.

The congestion of the pia mater produces delirium and stupor.

The congestion of the lungs gives labored breathing, and may go on to inflammation and consolidation of parts of the lungs.

The congestion of the pleura produces hydrothorax ; that of the stomach, vomiting ; that of the liver, enlargement of that organ ; that of the peritoneum, ascites ; that of the subcutaneous connective tissue, dropsy.

The congestion of the kidneys is attended with a diminished quantity of urine, which contains a little albumen and a few casts. If the urine is scanty for a long time the patients become anæmic and develop the symptoms of chronic uræmia.

2. The heart's action is feeble and rapid. There is not as much disposition to venous congestion, but the patients suffer from dyspnœa, they are very feeble, they are liable to attacks of syncope.

The emboli which give the most marked symptoms are those which become lodged in the cerebral arteries.

A large number of cases of acute endocarditis recover, but very few without permanent changes in the valves.

In some the valves, although rendered insufficient, undergo no further changes, and the patients do well.

In some the valves, after remaining unchanged for years, finally become the seat of chronic endocarditis.

In some the acute endocarditis passes into the chronic form, and the patients grow worse with but little interruption.

Treatment.—The principal objects of treatment are the cure of the rheumatism and the relief of the bad heart-action with the consequent disturbances of circulation. For the rheumatism the most efficient remedies are the salicylate of soda, the oil of wintergreen, the alkalies, antipyrine, and phenacetine.

If the heart's action is exaggerated, tumultuous, and irregular, with excessive venous congestion, we employ venesection, wet cups or leeches, purging or sweating. Chloral hydrate, amyl nitrite, opium, and potassium iodide are often of much service.

If the heart's action is feeble and rapid we need the cardiac stimulants—digitalis, convallaria, caffeine, and alcohol.

MALIGNANT ENDOCARDITIS.

Lesions.—Either the mitral, the aortic, or the tricuspid valve, the endocardium of the left auricle, or of the right ventricle may be inflamed. The inflamed endocardium is covered with a thin or thick layer of soft material, composed of fibrine and micrococci. The endocardium beneath is swollen, infiltrated with cells, often ulcerated. If the malignant endocarditis affects a heart already the seat of chronic endocarditis, the old lesions persist.

Portions of the soft matter on the inflamed endocardium often become detached and are lodged as emboli in the arteries of the brain, lungs, heart, spleen, and kidneys. These emboli are infectious and set up an inflammation of the surrounding tissues. So there may be complicating meningitis, pericarditis, bronchitis, pneumonia, or synovitis. The liver, spleen, and kidneys are swollen and their cells degenerated.

Causes.—Malignant endocarditis occurs with rheumatism, after childbirth, and from unknown causes.

Symptoms.—The disease begins with chills and a febrile movement. There is loss of appetite and some-

times vomiting ; the breathing is rapid. The heart's action may not be very much disturbed ; or it may be feeble, rapid, and irregular. The temperature continues high— 103° to 107° F.—but with occasional rapid falls. Cerebral symptoms are soon developed—restlessness, sleeplessness, delirium, and stupor. There is a heart-murmur if the endocarditis is sufficient to produce insufficiency, or roughening of the valves. The urine contains some albumen ; the joints may be inflamed ; there may be convulsions or paralyzes from embolism of the larger cerebral arteries. There may be bleeding from the mucous membranes and into the skin.

The patients pass into the typhoid condition and die, most at the end of seven to ten days, a few within three days, some not until after three to five weeks.

The whole picture is that of an infectious disease, with more or less pronounced evidence of the cardiac lesion.

CHRONIC ENDOCARDITIS.

Lesions.—The inflammation involves most frequently the mitral and aortic valves, less often the tricuspid and pulmonary valves and the endocardium of the ventricles and auricles. There are two forms of chronic endocarditis.

1. The endocardium is thick, dense ; there may be little beaded vegetations on its surface ; it may be infiltrated with the salts of lime. But its surface remains smooth, and thrombi are not formed on it. Such an endocarditis renders the valves rigid, insufficient, or stenosed.

2. The endocardium is thickened, there is a growth of

cells producing vegetations and ulcers, the surface of the endocardium is roughened, and thrombi are formed on it. In such an inflamed endocardium there may also be a deposition of the salts of lime.

When the valves are affected by either of these forms of endocarditis, they are rendered insufficient, or stenosed. These changes are followed by dilatation of the ventricles, by hypertrophy of their walls, and by disturbances of the circulation of the blood throughout the body.

With chronic endocarditis are often associated myocarditis, endarteritis, contraction of the arteries, emphysema, and chronic nephritis.

Causes.—Chronic endocarditis may succeed acute endocarditis, either immediately or after a long interval. It may be caused by rheumatism, gout, or syphilis. It very often is a primary lesion. It is rarely due to the rupture of a valve by external violence.

The symptoms depend on :

The endocarditis.

The dilatation and hypertrophy.

The character of the heart's action.

The presence of thrombi on the endocardium.

The myocarditis.

The endarteritis.

The contraction of the arteries.

The emphysema.

The chronic nephritis.

The general venous congestion.

1. The Endocarditis. The inflammation may last for months or years, cease to be active, and no further changes be produced in the valves ; or it may continue slowly and with intermissions up to the time of the pa-

tient's death, the valves becoming more and more damaged, and the inflammation extending to other valves ; or the inflammation may be more active, the endocardium more rapidly and profoundly changed, with a febrile movement and loss of nutrition.

The changes in the endocardium, due to the inflammation, may produce cardiac murmurs.

Aortic stenosis gives a murmur with the first sound, and during the first silence. The murmur is loudest at mid-sternum, opposite the third interspace. It is transmitted upward and to the right. It may be heard behind on the left side of the second, third, and fourth dorsal vertebræ. It may be loudest at the lower end of the sternum, or in the second interspace, on the right or left side. There may be well-marked aortic stenosis with no murmur.

Aortic insufficiency gives a murmur with the second sound, loudest at mid-sternum opposite the third interspace. It is transmitted down the sternum, and upward to the right. It may be loudest at the lower end of the sternum, in the left fourth interspace, or at the apex. It is a very constant murmur.

Mitral stenosis gives a presystolic, or systolic murmur, or both. The presystolic murmur is heard just before the first sound. It is loudest at the apex and is usually circumscribed. The systolic murmur is loudest at the apex and is transmitted to the left. The murmurs of mitral stenosis are very inconstant, and often absent altogether.

Mitral insufficiency gives a systolic murmur, loudest at the apex, and transmitted to the left. This murmur is nearly always a constant one.

Ventricular murmurs are produced by roughening of the ventricular endocardium, or thrombi in the ventricles. They are systolic, loudest at the apex, and circumscribed. They are not constant.

2. Dilatation and hypertrophy of the ventricles. Dilatation alone of the ventricles adds to the dangers of the endocarditis. Hypertrophy alone seems often to be of benefit. Hypertrophy and dilatation together, if not excessively developed, may also be of benefit. A heart which remains of normal size, or which becomes small, when associated with advanced valvular changes is apt to act badly.

Aortic stenosis is regularly followed by dilatation and hypertrophy, first of the left ventricle, then of the right.

Aortic insufficiency is followed by dilatation and hypertrophy of the left ventricle.

1 Mitral stenosis should be followed by dilatation and hypertrophy of the left auricle and the right ventricle. But there may be dilatation alone of either or of both ventricles ; or dilatation and hypertrophy of the left ventricle ; or the entire heart may be small.

Mitral insufficiency is followed by dilatation alone, or dilatation and hypertrophy of the left ventricle.

3. The characters of the heart's action. Disturbances of the heart's action seem to depend upon the changes in the valves and walls of the heart ; upon contraction of the arteries ; and upon unknown causes.

The heart's action may be rapid, slow, feeble, forcible, intermittent, irregular, or tumultuous. The contractions of the heart may be so feeble that there are more apex-beats than arterial pulsations.

Aortic and mitral stenosis, without contraction of the

arteries, gives a feeble and irregular pulse ; with contraction of the arteries, the pulse is tense and the heart-action often laboring. Aortic stenosis may give more ventricular contractions than heart-beats.

Aortic insufficiency gives an exaggerated heart-action with dilated arteries, full of blood, but soft.

Abnormal sensations referred to the heart seem to depend upon its disturbed action.

There may be actual pain, referred to the heart and the left shoulder ; the pain constant, produced by exertion, or paroxysmal. Or there are feelings of constriction, or of displacement of the heart.

4. Thrombi attached to the endocardium. These interfere with the heart's action. They may obstruct the orifices of the heart ; fragments of them lodged in the cerebral arteries produce convulsions, paralyses, and aphasia.

5. Myocarditis.—Interstitial myocarditis, degeneration of the heart-muscle, and disease of the coronary arteries are each one capable of rendering the heart's action feeble and irregular ; of producing attacks of angina pectoris ; and of causing sudden death.

6. Chronic endarteritis affecting many of the arteries adds to the difficulties of the circulation. If it involves the cerebral arteries the characteristic symptoms of cerebral endarteritis may be developed.

7. Pulmonary emphysema is a frequent complication, and adds much to the dangers of the endocarditis.

8. Venous congestion takes a very large share in producing the symptoms of chronic endocarditis. It is due to the valvular lesions, the dilatation of the ventricles, the disturbed heart-action, the contracted arteries, and the complicating emphysema or endarteritis.

Congestion of the pia mater produces delirium, stupor, and convulsions.

Congestion of the lungs produces chronic bronchitis, hæmoptyses, the pneumonia of heart disease, œdema of the lungs, and hydrothorax.

Congestion of the stomach produces pain, gastric dyspepsia, vomiting, and vomiting of blood.

Congestion of the small intestine produces loss of flesh and strength.

The general congestion of the body gives subcutaneous dropsy and dropsy of the serous cavities.

The kidneys may be the seat of chronic congestion, of chronic degeneration, or of chronic nephritis.

With chronic congestion of the kidney the quantity of urine is diminished, its specific gravity is high, there is little or no albumen, the quantity of urea to the ounce of urine is sufficient. This change in the kidney seems to add but little to the symptoms.

The chronic congestion, if protracted, is succeeded by a chronic nephritis. The specific gravity of the urine falls, the quantity of urea is diminished, and albumen and casts make their appearance.

With chronic degeneration of the kidney the quantity of the urine is diminished, the specific gravity and the proportion of urea are unchanged, there is a moderate quantity of albumen and a few casts. If the excretion of urine is scanty for a long time the patients may pass into the condition of chronic uræmia.

The chronic nephritis is diffuse and most frequently follows one of two types.

1. There is an abundant exudation of serum from the vessels of the kidneys. The urine is of low specific

gravity and contains large quantities of albumen. The patients are anæmic, dropsical, and suffer from chronic uræmia.

2. There is little or no exudation from the blood-vessels of the kidneys, but there is an extensive growth of interstitial connective tissue. The urine is of low specific gravity, contains little urea, and little or no albumen. The patients are liable to attacks of acute uræmia, with contracted arteries.

Course of the Disease.—The disease may succeed one or more attacks of acute endocarditis, or it may be chronic from the outset.

In the chronic cases the symptoms are usually not developed until the disease has existed for some time. Then first one or two symptoms appear and others are added later.

The first symptom may be :

Dyspnœa on exertion.

Anæmia and emaciation.

Cough and expectoration.

Cardiac palpitation and pain.

Stomach symptoms.

Dropsy.

Contraction of the arteries.

It sometimes happens that the patients go on for a long time with an endocarditis, but without symptoms. Then suddenly, with a pericarditis, a pleurisy, a bronchitis, or without discoverable cause, they develop a most distressing and urgent dyspnœa with contracted arteries. This dyspnœa continues and the patients die within a few days or weeks.

The endocarditis may stop at any time. It may go

on slowly, or quickly ; it may be complicated by the diseases already mentioned.

The bad cases die with dyspnœa ; or feeble and emaciated ; or with dropsy ; or with cerebral symptoms ; or they die suddenly.

The prognosis depends upon :

The extent and activity of the endocarditis.

The condition of the ventricles.

The character of the heart's action.

The presence or absence of contraction of the arteries.

Treatment.—The principal indications for treatment are to check the progress of the endocarditis, to regulate the action of the heart and the condition of the arteries, and to alleviate symptoms.

To check the progress of the endocarditis we put the patient on a diet of fats, meat, fruits, and vegetables ; we prohibit the use of alcohol and tobacco, and we insist on a life passed in the open air.

To regulate the action of the heart and the condition of the arteries. If the arteries are contracted we employ nitrite of amyl, nitro-glycerine, potassium iodide, chloral hydrate, or opium.

If the heart's action is feeble we use digitalis, strophanthus, caffeine, or convallaria. If the heart's action is too forcible we use continuous cold over the heart, a nitric-acid issue, aconite, veratrum viride, or barium chloride.

With aortic, or mitral, stenosis and a feeble heart it may be necessary to confine the patients to bed.

With many forms of endocarditis the patients do best if they are allowed to follow their ordinary mode of life, avoiding sudden and violent exertion.

In a certain number of cases very excellent results are obtained by regulated hill-climbing.

HYPERTROPHY AND DILATATION OF THE HEART WITHOUT ENDOCARDITIS.

I. From excessive and prolonged muscular exertion.

The left ventricle alone, or both ventricles, are hypertrophied or dilated, or both dilated and hypertrophied.

To produce these changes in the heart the muscular exertion must be excessive and prolonged, so we find them most frequently in soldiers, athletes, and laborers.

In the milder cases there is only hypertrophy of the left ventricle. The patient has the feelings of pain, or constriction, or displacement of the heart, with rapid heart-action.

These patients usually do well. It is only necessary to make them lead a regular life and sometimes to take digitalis.

In the more severe cases there is dilatation alone, or dilatation with hypertrophy. There are pain over the heart, rapid and feeble heart-action, dyspnœa on exertion, and loss of flesh and strength. These cases are always serious, and not all of them recover.

It is necessary to keep the patients in bed, and to use cardiac stimulants.

II. From disease of the lungs.

Emphysema, chronic phthisis, interstitial pneumonia, pleurisy, and deformities of the chest may produce dilatation, first of the right ventricle, and afterward of the left.

III. Exophthalmic goitre, or prolonged palpitation of

the heart, may be followed by hypertrophy of the left ventricle.

IV. Acute or chronic nephritis, chronic inflammation of the aorta, and congenital narrowing of the aorta may produce hypertrophy of the left ventricle.

V. Simple anæmia may be attended with temporary dilatation and hypertrophy of the left ventricle.

VI. There is an important group of cases of dilatation of the ventricles without hypertrophy, of which the causation is very obscure.

Usually both ventricles are dilated, but sometimes only the left. The valves are normal, but their orifices may be so large that they are insufficient. The walls of the ventricles are normal, or the muscular fibres are degenerated, or there is a growth of cells between the fibres. Thrombi may be formed in the cavities of the ventricles. Venous congestion of the viscera is soon established, dropsy somewhat later.

Of the causes of this heart-lesion we are ignorant. It is often enough developed in young and healthy adults. In some cases there is a distinct history of sudden muscular exertion ; in others, no exciting cause can be discovered.

In the more acute cases the invasion of the symptoms is sudden.

The patients complain of pain in the epigastrium, they become unconscious, and they vomit ; or the first symptom is urgent cardiac dyspnoea. After this the heart's action is rapid, irregular, and feeble. The physical signs are those of dilatation of the heart. There is constant and distressing dyspnoea, there may be cough and hæmoptyses, the patients lose flesh and strength, and die at the end of a few weeks.

In the more chronic cases the invasion is less sudden and decided. The patients suffer from rapid, feeble, and irregular heart-action, dyspnœa, cough, and hæmoptyses, marked dropsy, and loss of flesh and strength. They may live for several months.

The treatment of these cases is eminently unsatisfactory. We are unable to improve the action of the heart, and the best that we can do is to palliate the patient's sufferings with opium.

THE FATTY HEART.

1. There is an accumulation of fat beneath the pericardium. This occurs in persons who also have accumulations of fat in other parts of the body.

The heart's action is often feeble, and there is dyspnœa on exertion. It is difficult to tell whether the cardiac symptoms are due to the fat on the heart or to the general condition of the patient.

The indications for treatment are by a regulated diet and systematic exercise to remove the fat.

2. There is a fatty degeneration of the muscular fibres of the wall of the heart.

The heart remains of its normal size, or becomes somewhat smaller. Its walls are soft and lighter colored. The degeneration may involve the walls of both ventricles, or of one, or of part of one. The degeneration may advance so far at some one point that the wall of the ventricle will be ruptured.

Causes.—This change in the heart may be secondary to profound anæmia, to long and wasting diseases, to the severe infectious diseases, and to poisoning by phosphorus. It occurs also as a primary disease, and is then es-

pecially common in males between the ages of forty and seventy.

Symptoms.—Physical signs: The heart is of normal size, or diminished. The impulse is feeble; the first sound is short and feeble; the action may be regular, irregular, fast or slow. The systolic murmur at the apex, or at the second left interspace, which occurs without valvular lesions, may be present.

There may be pain, referred to the heart, either continuous or spasmodic; or attacks of angina pectoris.

There may be attacks of syncope, dyspnœa on exertion, or constant dyspnœa. Cerebral symptoms: Vertigo, convulsions, loss of consciousness, or coma may be developed. There may be gradual loss of flesh and strength. Dropsy is not a frequent symptom.

Some of the patients never give any symptoms of their cardiac lesion, and die of some other disease.

Some give no symptoms until they have a fatal attack of angina, of syncope, of coma, or of rupture of the heart.

Some give the characteristic symptoms of the disease for months or years.

Treatment.—The best that can be done is the regulation of the diet and mode of life, and the use of cardiac stimulants.

RUPTURE OF THE HEART.

Lesions.—Ruptures of the heart are most common in the anterior wall of the left ventricle near the apex. They also occur in the wall of the right ventricle, the right auricle, the septum between the ventricles and the papillary muscles. The rupture is small, direct, or indi-

rect. The rupture is due to fatty degeneration of the muscle, or to occlusion of one of the branches of the coronary artery.

Symptoms.—The rupture may occur during exertion, or while the patient is perfectly quiet.

If the rupture is direct death is almost instantaneous. If it is indirect the patients may live for hours or days. They have sudden pain in the heart, vomiting, dyspnoea, prostration, irregular and feeble heart-action.

THE NEUROSES OF THE HEART.

PALPITATION.

There is no structural change in the heart, except that in the long-continued cases the left ventricle may be hypertrophied.

The heart's action is too frequent, and may be irregular. There may be an apparent exaggeration of the heart's action, but yet it is a feeble heart.

The patients suffer from the consciousness of the heart's action, from abnormal cardiac sensations, from vertigo, headache, fulness of the head, syncope, sleeplessness, loss of flesh and strength, and a variety of nervous phenomena.

Many of the cases are mild and easily relieved, but some are very serious.

Causes.—In some of the cases there is apparently a cause for the palpitation—gastric and hepatic dyspepsia, an abnormal mode of life, the abuse of tobacco and of tea, and anæmia. In other cases no cause can be discovered.

Treatment.—The indications for treatment are to ob-

tain control over the patient, to remove the causes of the disease, to regulate the mode of life, and to use cardiac stimulants.

ANGINA PECTORIS.

The disease is characterized by pain, oppression, a sense of impending death, abnormal action of the heart, or contraction of the arteries.

Lesions.—There may be inflammation of the coronary arteries, fatty degeneration of the heart, inflammation of the aorta, chronic endocarditis, changes in the cardiac plexus of nerves, or no discoverable lesions.

Causes.—The disease is especially common in males, among the better classes, and in persons over fifty years of age.

Symptoms.—The symptoms occur in attacks. Between the attacks the patients are perfectly well, or they suffer from the symptoms of the disease of the coronary arteries; of the fatty heart; of the diseased aorta; or of the chronic endocarditis.

During the attack the patients have: Pain and tenderness, referred to the precordial part of the lower sternal region, or to the middle or upper sternal region. The pain radiates to the mid-dorsal spine; to the left, or to both sides of the neck and the occiput; to the left shoulder and arm, to both arms, to the right arm alone, to the left leg, or to both arms and the left leg. With the pain there may be feelings of numbness and tingling.

A feeling of cardiac oppression and of impending death, which they describe very vividly.

A pulse of high tension, or a feeble and irregular pulse.

Dyspnœa in some of the cases, but not in all.

Vomiting, flatulence, eructations of gas. An unimpaired intelligence, or slight convulsive movements, or general convulsions, or loss of consciousness.

An anxious face, with a skin cold and bathed in perspiration.

The attacks usually come on suddenly and without warning. They may, however, be preceded by abnormal cardiac sensations, or be excited by certain muscular movements, or occur at regular intervals.

Each attack may last for a few minutes, or for several hours. A patient may have only a single attack, from which he recovers, or during which he dies. He may have a number of attacks any one of which may prove fatal ; or after a number of attacks there may be no further recurrence.

The prognosis of the disease is very serious, but yet there is a considerable number of persons who suffer from one or more attacks and entirely recover.

Treatment.—For purposes of treatment we divide the cases into two classes : Those in which, during the attack, the arteries are contracted, with a pulse of high tension ; and those in which, without any such contraction of the arteries the heart's action is feeble and irregular.

The attacks with contraction of the arteries can usually be relieved by inhalations of nitrite of amyl, hypodermic injections of morphine, and the internal use of nitroglycerine. The attacks without contraction of the arteries are very much more difficult to manage.

During the intervals between the attacks we try to improve the general health of the patient in every possible way.

PSEUDO-ANGINA PECTORIS.

We use this name to designate a somewhat irregular group of cases, which present the common feature of attacks of disturbance of the action of the heart.

Lesions.—There may be chronic inflammation of the coronary arteries, fatty degeneration of the heart, inflammation of the arch of the aorta, or no discoverable lesion.

Symptoms.—Pain may be absent altogether. When present it is not as severe as the pain of true angina, but is referred to the same regions.

The patients may be much alarmed about themselves, but do not have the feeling of impending death.

The heart's action is feeble and irregular, although sometimes it may seem to be exaggerated.

The breathing may be hurried and labored. There may be vomiting and pain in the stomach.

The attacks come on suddenly, they last for minutes, days, or weeks. In the longer attacks the symptoms are not continuous.

The patient may die in any of the attacks; or after one or more such attacks there may be no further recurrence.

Treatment.—During the attacks we use cardiac stimulants; between the attacks we try to improve the general health.

EXOPHTHALMIC GOITRES.

Basedow's disease. Graves' disease.

The cases are characterized by rapid heart-action, enlargement of the thyroid gland, and protrusion of the eyeballs.

Lesions.—After death the thyroid gland is found simply hypertrophied, there is nothing in the orbits, the left ventricle of the heart may be hypertrophied. In a few cases in the ganglia of the cervical sympathetic there have been found degeneration of the nerve-cells and a new growth of connective tissue.

Causes.—The disease is much more common in women than in men, and especially during early adult life.

Symptoms.—The symptoms are usually developed slowly, but exceptionally within a few days. The first symptom is regularly the increased frequency of the heart's action, but without irregularity or feebleness. Then comes the moderate and symmetrical enlargement of the thyroid, sometimes with dilatation of its veins.

The enlargement of the thyroid may precede the palpitation; it may be absent altogether.

Next comes the exophthalmos. It may, however, precede the goitre; it may be the first symptom; it may be absent altogether.

After a time there may be developed hypertrophy of the left ventricle of the heart.

Besides the characteristic symptoms of the disease, there may be added a variety of nervous phenomena, dyspnoea, anæmia, and loss of flesh and strength.

The disease is apt to last for years; it is not of itself fatal. Some of the patients recover altogether, some only in part.

Treatment.—The indications are to improve the general health of the patient in every way; to relieve the anæmia; to give the cardiac stimulants and the iodides of potassium and sodium; and to use the galvanic current over the cervical sympathetic.

ANEURISM OF THE AORTA.

Lesions.—There is a chronic inflammation of the wall of the aorta ; by this the wall of the aorta is thickened, or thinned, or ulcerated. The thickened portions may undergo fatty degeneration, or become calcified. As a result of these changes there may be rupture of the aorta, diffuse dilatations, or sacculated dilatations. So there are formed dissecting, fusiform, and sacculated aneurisms.

DISSECTING ANEURISMS.

Peacock describes 19 cases. They occurred in persons between the ages of twenty-four and eighty-four years. Crisp describes 21 cases. They occurred between the ages of twenty-four and ninety years. Seven were in males, fourteen in females. Sibson describes 52 cases.

The rupture of the inner coat is usually in the ascending portion of the arch, near the valves. The blood finds its way between the coats of the aorta, and then ruptures through the outer coat within the pericardium, or lower down.

The patients usually have no symptoms until the rupture of the aorta. Then they die suddenly, or they live for hours or days with great prostration and feeble heart action.

FUSIFORM AND SACCULATED ANEURISMS.

Fusiform and sacculated aneurisms occur either separately or together.

The fusiform dilatations may involve the arch alone, or the whole aorta.

The sacculated aneurisms have a small or large opening. The sacs are small or large; they are single or multiple.

Causes.—These aneurisms are especially common in males between the ages of thirty and fifty years; in persons who have constitutional syphilis; in persons who have chronic inflammation of the aorta, and whose occupation involves severe muscular exertion.

Situation.—Sibson gives the situation of 860 cases of aneurism of the aorta as follows:

Of the arch of the aorta, 480.

At the sinuses of Valsalva, 87.

Of the ascending portion of the arch, 141.

Of the transverse portion of the arch, 120.

Of the ascending and transverse portions of the arch together, 112.

Of the descending portion of the arch, 72.

Of the transverse and descending portions of the arch together, 20.

Of the whole arch, 28.

Of the thoracic aorta, 71.

Of the abdominal aorta at the cœliac axis, 131.

Of the lower part of the abdominal aorta, 26.

Symptoms.—These are due to the presence of the aneurismal tumor, its pressure on the surrounding parts, and its effect on the circulation of the blood.

The tumor, if situated within the thorax, gives dullness on percussion, the feeling of a pulsating tumor, a double murmur, and a thrill. If it is situated within the abdomen, it gives the feeling of a pulsating tumor, a murmur, and a thrill.

The pressure of the tumor is on :

The descending vena cava, with venous congestion of the head and neck.

The trachea and large bronchi, with cough, dyspnœa, stridulous cough, voice, and breathing, and tubular breathing over the lungs.

The pulmonary artery, the right auricle, and the upper part of the ventricles, with disturbances of the circulation.

The œsophagus, with difficulty in swallowing.

The recurrent laryngeal nerve, with paralysis of one of the vocal chords.

The brachial plexus of nerves, with pain.

The vertebræ, with pain.

Any sacculated aneurism is liable to rupture.

ANEURISMS AT THE SINUSES OF VALSALVA.

These aneurisms are sacculated, and are most frequently given off from the right coronary sinus.

Small aneurisms in this situation give no symptoms, unless they rupture within the pericardium and cause sudden death.

Larger aneurisms may compress and erode the pulmonary artery, the right auricle, or the right ventricle.

There is an area of dullness, or a pulsating tumor, either to the right or left of the sternum, at about the third interspace.

There is a murmur in 34 per cent. of the cases.

Dyspnœa in 38 per cent.

Cough in 24 per cent.

Pain in 21 per cent.

An enlarged heart in 29 per cent.

Rupture of the aneurism within the pericardium in 48 per cent.

In the pulmonary artery in 13.5 per cent.

In the right auricle in 8.5 per cent.

In the right ventricle in 5 per cent.

In the left ventricle in 5 per cent.

ANEURISMS OF THE ASCENDING PORTION OF THE ARCH.

These aneurisms are sacculated in 51 per cent., fusiform in 37 per cent. of the cases.

The sacs are given off from the anterior wall, the right wall, the left wall, and the posterior wall.

They compress the right lung, the left lung, the right bronchus, the left bronchus, the pulmonary artery, the descending vena cava, the trachea, or the œsophagus. There is an area of dulness, or a pulsating tumor, beneath and to the right of the sternum, at about the second interspace.

A murmur is present in 23 per cent. of the cases; dyspnœa in 51 per cent.; cough in 36 per cent.; stridor in 4 per cent.; pain in the chest in 29 per cent.; in the neck and shoulders in 10 per cent.; in the right arm in 2 per cent.

About 11 per cent. of the patients are in good health up to the time of their death.

The aneurisms rupture in 57 per cent. of the cases:

within the pericardium, externally, into the descending vena cava, into the right lung, the pulmonary artery, the left pleura, the right bronchus, the left lung, the right pleura, the trachea, or the right auricle.

ANEURISMS OF THE TRANSVERSE PORTION OF THE ARCH OF THE AORTA.

These aneurisms are most frequently sacculated. The larger number are given off from the posterior wall of the aorta; a smaller number from the anterior, or upper wall.

Those given off from the posterior wall soon compress the trachea or bronchi, the œsophagus, and the recurrent laryngeal nerve. The patients very soon have dyspnœa, cough, stridor, paralysis of one of the vocal chords, tubular breathing over one or both lungs. But the physical signs of the tumor are absent or obscure until it reaches a large size. These aneurisms are apt to rupture into a bronchus, the trachea, or the œsophagus.

The aneurisms given off from the anterior and upper wall soon give the physical signs of a tumor; they make a difference in the pulsation of the carotid and radial arteries. They must attain some size before they exert much pressure.

ANEURISMS OF BOTH THE ASCENDING AND TRANSVERSE PORTIONS OF THE ARCH OF THE AORTA.

These are very often fusiform aneurisms. The physical signs of the tumor are soon evident. The pressure is on the anterior wall of the chest, the large vessels, the

bronchi or trachea, the recurrent laryngeal nerve, and the œsophagus.

ANEURISMS OF THE DESCENDING PORTION OF THE ARCH OF THE AORTA.

These aneurisins are most frequently sacculated. They are given off from the posterior and left wall of the aorta. The vertebræ are eroded in 42 per cent. of the cases. The aneurism may compress the left bronchus, the œsophagus, the left lung, the trachea, the right lung, or the ribs in front.

The principal symptoms are the pain from the erosion of the vertebræ, and the cough and dyspnœa from the pressure on the bronchi, the trachea, and the lungs.

ANEURISMS OF THE THORACIC AORTA.

These are sacculated or fusiform in the proportion of thirty-five to twenty-eight.

They are given off from the posterior wall of the aorta. Most of them erode the vertebræ. They may compress the œsophagus and the left lung, and may form a tumor in the back.

The most marked symptom is the pain due to the erosion of the vertebræ.

ANEURISMS OF THE ABDOMINAL AORTA.

These are usually sacculated. The larger number are situated near the cœliac axis. They are given off with about equal frequency from the anterior and the posterior walls of the aorta.

Those on the posterior wall very early erode the vertebræ.

The anterior aneurisms give pain referred to the epigastrium and the anterior portion of the abdomen. The tumor is easily made out by palpation.

The posterior aneurisms give pain referred to the vertebræ, and radiating downward. The tumor is often obscure.

These aneurisms rupture into the peritoneal cavity, behind the peritoneum, into the mæsenteric, and into the right or left pleural cavity.

There may be incomplete or complete obstruction of the aorta by thrombi extending from the aneurismal sac.

Treatment.—The medical treatment of aneurism of the aorta consists in determining for each patient, experimentally, whether he is better for rest, or for regulated exercise; and in the administration of the iodide of potash. The iodide is given in doses of from five to twenty grains, three or four times a day. In favorable cases the aneurism is diminished in size, the symptoms are alleviated, and life is prolonged.

THE ŒSOPHAGUS.

STENOSIS.

1. Congenital stenosis of the œsophagus is rare. It is most frequently seen in children, who die early. Exceptionally, the patients survive to adult life, but with constantly increasing difficulty in swallowing.

2. Stenosis by compression is due to the pressure of aneurisms, of tumors of the thyroid, and of tumors of the neck and the thorax. The only symptom is incomplete difficulty in swallowing.

3. Stenosis from tumors of the posterior wall of the pharynx, either polypoid or diffuse.

4. Cicatricial stenosis from the contraction following the lesions inflicted by irritant poisons. These require surgical treatment.

5. Stenosis due to syphilitic inflammation of the wall of the œsophagus.

6. Carcinoma of the œsophagus.

The new growth, of flat-celled epithelial type, begins at some part of the wall of the œsophagus, and soon surrounds it. There are masses of new growth, and deep ulcers; either may be the predominant feature. The new growth involves only one or two inches of the œsophagus, or a much larger portion. The lower half of the œsophagus is the portion most frequently in-

volved. The growth may remain confined to the wall of the œsophagus, or may extend to the trachea, bronchi, lungs, pleura, pericardium, and vertebræ. Metastatic tumors may be formed in the lymphatic glands, lungs, and liver. A cancer of the stomach may be developed at the same time with a cancer of the œsophagus.

The disease occurs most frequently in males over forty years of age.

The first symptom is difficulty in swallowing. This begins with difficulty in swallowing large pieces of solid food, then smaller pieces cannot be swallowed, and, finally, not even liquids. The progress of the dysphagia is gradual, and often intermittent.

The next symptom is pain, referred to the upper end of the sternum.

It is not until the tumor has existed for some time, and the difficulty in swallowing is considerable, that there is much change in the nutrition of the patients. But after they have once begun to emaciate they get worse very rapidly.

Life may be prolonged for a few weeks by passing tubes through the œsophagus into the stomach, and by the use of rectal alimentation.

7. Spasmodic stricture occurs in young and old persons, in both males and females. There is sometimes a history of hysteria, sometimes one of sudden choking.

The one symptom is difficulty in swallowing, sometimes attended with pain.

The dysphagia may be complete, or partial. It occurs in attacks which last for minutes, days, weeks, or months; and which are repeated at short or long intervals.

The best treatment is the passage of large tubes through the œsophagus, at regular intervals.

DILATATION.

1. Fusiform dilatations of the œsophagus are secondary to stenosis, or occur without discoverable cause. They involve a part, or the entire length, of the œsophagus. The wall of the dilated œsophagus is often thickened.

The symptoms are difficulty in swallowing, vomiting, regurgitation of food, and rumination.

The treatment consists in the dilatation of the stenosis and the feeding the patients with the stomach-tube.

2. Sacculated dilatations are of two kinds.

(a) Small sacs are formed in the lower part of the posterior wall of the pharynx, which afterward become larger.

They are more common in males than in females, and belong to adult life.

How it is that these sacs begin to be formed, is not well understood. But as soon as they have reached such a size that the food can enter and remain in them, they rapidly enlarge behind the œsophagus and compress it.

There is difficulty in swallowing, at first slight, but increasing with the pressure of the sac on the œsophagus.

There is regurgitation of the food from the sac, rumination, and vomiting. There may be a tumor in the neck which can be emptied by pressure.

The only treatment which has thus far been employed is to feed the patients with the stomach-tube.

(*b*) Small sacs are given off from the anterior wall of the œsophagus, at about its middle. They seem to be due to an inflammation of the bronchial glands, an extension of this inflammation to the wall of the œsophagus, and traction.

These sacs give no œsophageal symptoms, but they may perforate and so give rise to inflammatory processes in the mediastinum.

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THE STOMACH.

Digestion is effected by the stomach, the intestines, the liver, and the pancreas.

The stomach receives the food, it keeps it in motion, it digests certain portions of it, and after a certain time it expels all of it, digested and undigested, into the intestine.

Gastric digestion is effected by the gastric juice, which changes albumen, gluten, fibrine, caseine, and gelatine into peptones. The starches, sugars, and fats are digested in the intestine.

To assist the action of the gastric juice the muscular coat of the stomach must move the food, and at the proper time must empty the stomach.

GASTRIC DYSPEPSIA.

This name is ordinarily used to designate disturbances of the digestive function of the stomach, and abnormal sensations referred to that organ.

Causes.—I. Food. The introduction into the stomach of food in such quantity and of such quality that it cannot be digested causes such food to act as an irritant.

We find that the distention of the stomach with food may stop gastric digestion altogether, and the patient will only be relieved by vomiting.

We find also that the habitual presence in the stomach

of undigested food may produce catarrhal gastritis and dilatation of the stomach. The passage of the same undigested food into the intestine causes trouble there.

2. Abnormal gastric juice. The normal production of gastric juice may be interfered with by a variety of mental and nervous influences, by impaired health, by old age, and by diseases of the mucous membrane.

3. Diseases of the mucous membrane of the stomach—inflammation, degeneration, ulcers, and new growths.

4. Changes in the muscular coat of the stomach which interfere with the movement and expulsion of the food, and permit of dilatation.

5. Climate and mode of life.

Symptoms.—The appetite may be diminished, there may be aversion to food ; the appetite may be unnaturally increased, or it may be perverted.

Pain is referred to the epigastrium, the left hypochondrium, the præcordium, the sternum, or the left shoulder-blade. The pain may be continuous, or may come on in attacks.

Instead of the pains there may be feelings of oppression, of emptiness, of burning, of gnawing, etc.

All these abnormal sensations may occur when the stomach is full, or when it is empty. They are not in any proportion to the actual disturbance of digestion.

Nausea is a common symptom. It is felt in the early morning, at the sight of food, or after eating.

There may be vomiting of food, of mucus, of serum, or of blood. Large quantities of food, which the stomach refuses to digest, are regularly vomited. Inflammation and congestion of the mucous membrane, an undue

production of mucus and serum, the retention of food in the stomach, stenosis of the pylorus, ulcers, and new growths, are all causes of vomiting. Vomiting may also be due to causes remote from the stomach—constipation, uterine disease, or anæmia.

The name of “water brash” is given to the eructation of sour fluid from an empty stomach.

Flatulence is rather an intestinal than a gastric symptom. But one of its causes is the passage into the intestine of portions of food which ought to have been digested in the stomach.

Constipation seems, in some persons, to be entirely due to disorders of the stomach.

Headache, facial neuralgia, fulness of the head, vertigo; pains over the heart, lungs, and abdomen; feelings of palpitation, oppression, and dyspnœa occur in persons who, besides their gastric dyspepsia, also suffer from disturbances of the functions of the liver and intestine.

ACUTE CATARRHAL GASTRITIS.

Lesions.—There is an acute catarrhal inflammation of the mucous membrane of the stomach, with congestion and an increased production of mucus. The inflammation may involve only the pyloric end of the stomach, or the entire mucous membrane.

Causes.—The disease occurs at all ages; it is more common in summer than in winter. It may be due to improper food, or to alcohol.

Symptoms.—The prominent symptoms are pain, vomiting, a febrile movement, prostration, and constipation.

In infants the vomiting is incessant, the rise of tem-

perature often well marked, the prostration considerable. The gastritis may be followed by diarrhœa.

If the attack occurs in hot weather, attention should be given to keeping the child as cool as possible. The food should consist of cream and water, of koumyss, of beef juice, or of scraped meat. For drugs, calomel, oxalate of cerium, and bicarbonate of soda are the most useful.

In older children the symptoms may be the same as in infants. Or the child may complain of general malaise, headache, loss of appetite, nausea, constipation, and a constant pain in the abdomen. Vomiting is not a prominent symptom. Fever is present or absent. The patients lose flesh and strength. The attacks last for about a week, and may be followed by jaundice.

The patients should be fed with small quantities of cream and water, with milk, koumyss, beef juice, or scraped beef.

Calomel, oxalate of cerium, bicarbonate of soda, and opium may be of service. In some cases there is an advantage in using ice-water enemata.

In adults, the most common form of the disease is that in which incessant vomiting, prostration, and constipation are the prominent symptoms. For these patients small hypodermics of morphine are perhaps the most efficient remedy. In some of these cases there is added a febrile movement; in some the prostration and feeble heart action are so extreme that stimulants are required.

In other adults the disease follows the type of that seen in children, without much vomiting, but lasting for a longer time. In these patients ice-water enemata are of service.

CHRONIC CATARRHAL GASTRITIS.

Lesions.—In many cases the only evidence of the inflammation is the production of an increased quantity of mucus, which coats the inner surface of the stomach. In other cases there is added a degeneration of the cells of the peptic glands. In still others there is a growth of connective tissue between the gastric tubules, with atrophy of the tubules. Or there may be hypertrophy of the connective tissue and muscular coats of the stomach, with stenosis of the pylorus. The inflamed stomach may remain of its natural size, or be contracted, or dilated.

When we say that a patient has chronic catarrhal gastritis, therefore, we mean either :

That the mucous glands are constantly producing too much mucus.

Or, that, in addition, the function of the peptic glands is disturbed.

Or, that the structure of the mucous membrane is so impaired by disease that gastric digestion is seriously interfered with.

Or, that, from stenosis of the pylorus, or relaxation of the muscular coat, food is retained in the stomach.

Or, that the stomach is dilated, or contracted.

Causes.—Cardiac disease, pulmonary emphysema, and cirrhosis of the liver produce, first, chronic congestion, and then chronic gastritis. Chronic Bright's disease, phthisis, gout, rheumatism, and chronic alcoholism, are often attended with gastritis. Climate and mode of life seem to be important factors in the production of the disease. The infectious diseases, especially typhoid fever, may be followed by chronic gastritis.

The *Symptoms* are : Pain or a feeling of discomfort in the stomach while gastric digestion is going on. Attacks of pain, often relieved by vomiting, due to the presence in the stomach of portions of food which irritate it. The peculiar feeling called "heart-burn." Attacks of neuralgic pain for which it is difficult to account.

Nausea, early in the morning or nearly constant. Except for the nausea, the appetite is apt to be fairly good.

Vomiting of food : To remove the portions of food which irritate the stomach ; of large quantities of brownish fluid ; of small quantities of pure mucus ; of food which has not been able to escape through the pylorus ; of small quantities of very acid fluid ; of blood.

Retention of food in the stomach, with or without dilatation of the stomach, or stenosis of the pylorus.

Constipation, or diarrhoea. Headache in attacks. Loss of flesh and of strength, anæmia, catarrhal pharyngitis, swelling of the tongue, abnormal sensations referred to the mouth, throat, and tongue.

With many cases of chronic gastritis there are also developed disturbances of the functions of the liver and of the intestines.

The course of the disease is prolonged, with exacerbations and intermissions. The symptoms, therefore, are not continuous, but come on in attacks. As the disease progresses these attacks become more frequent, more severe, and of longer duration.

In some patients the disease never becomes more than an annoyance. In some the loss of flesh and strength and the anæmia are so pronounced, that the patient is unable to work. The addition of disturbance of the

functions of the liver to the gastritis are often attended with distressing nervous and mental phenomena. The pain may be so severe and continuous that the patient's life is rendered miserable.

Treatment.—The mild cases of chronic gastritis are to be treated by regulating the diet and the mode of life. They should take three regular meals a day, composed of meat, vegetables, fruit, and bread; no pepsine, no peptonoids, no health foods. They should give up the use of tobacco, tea, and liquor. They should have every day an hour during which they take exercise sufficiently active to produce a thorough perspiration.

The more severe cases are best treated by the use of the stomach-tube, and the washing out of the stomach with hot water. The washing the stomach is done at first once a day, then every other day, and then at longer intervals. The patient at first only takes one solid meal a day of meat, bread, and water; during the rest of the twenty-four hours he takes only milk. After he has begun to improve, other articles of food are added, until he takes three ordinary meals a day.

If it is not possible to use the stomach-tube, the treatment of these cases is much less satisfactory. They can be improved for a time, but not cured. It is necessary for weeks or months to put them on an exclusive diet, either milk alone, or beef and hot water alone. The bowels must be kept open with laxatives or enemata. There may be an advantage in giving alkalies, or the mineral acids, bismuth, iodoform, carbolic acid, etc

SUPPURATIVE GASTRITIS.

Lesions.—There is a suppurative inflammation beginning in the connective-tissue coat of the stomach, and extending to the other coats. This inflammation may be circumscribed, with the formation of an abscess; or diffuse, with a purulent infiltration of the entire connective-tissue coat. The abscesses may rupture into the cavity of the stomach. The inflammation of the peritoneal coat may give rise to a general peritonitis.

Causes.—The disease belongs to adult life. It is more common in males than in females. There is, in some cases a history of the over-eating of indigestible food.

Symptoms.—The formation of the abscesses in the wall of the stomach is attended with pain in the epigastric region, vomiting, a febrile movement, and the formation of a tumor. If the abscess ruptures into the stomach, the pus may be vomited. The peritonitis often remains localized. The disease may run an acute or a chronic course.

The diffuse suppurative inflammation of the wall of the stomach runs an acute course, terminating fatally in from three to eighteen days. The patient is suddenly attacked with vomiting, pain, and tenderness over the stomach, fever, and great prostration. The symptoms of general peritonitis are soon added, and the patients rapidly get worse.

Treatment.—The best that we can do seems to be to alleviate the patient's symptoms by the liberal use of opium.

ULCER OF THE STOMACH.

Lesions.—Ulcers of the stomach are usually single, but two or more ulcers may be formed at the same time, or successively.

Welch, from a collection of 793 cases, gives the position of such ulcers as follows :

On the lesser curvature, 288.

On the posterior wall, 235.

At the pylorus, 95.

On the anterior wall, 69.

At the œsophageal end, 50.

At the fundus, 29.

On the greater curvature, 27.

The ordinary diameter of these ulcers is from half an inch to two inches, but some are very small, and some are much larger. Two or more ulcers may become joined, and so form a large ulcerated area.

The ulcers are round or oval, largest in the glandular coat. They may destroy only the glandular coat, or the entire thickness of the wall of the stomach. The edges of the ulcers are clean-cut, and their floors smooth ; but sometimes the edges are much thickened, and the floor may be formed by tissues which have become adherent. The edges and the floor are formed of amorphous granular matter, or of connective tissue.

If the patients recover, the ulcers cicatrize either with or without deformity of the stomach.

If the ulcers perforate the wall of the stomach, this perforation may be large and sudden, with the escape of the contents of the stomach into the abdominal cavity ; or the perforation may be small, and set up a localized

peritonitis; or the opening may be entirely closed by adhesions.

The ulcer may erode either the arteries or the veins of the stomach. With an ulcer there is usually more or less chronic gastritis.

Causes.—These ulcers are said to be twice as common in women as in men. They have been observed at nearly every age, but seven-tenths of them are in people between twenty and forty years of age.

The ordinary explanation of the way in which these ulcers are formed is, that by embolism, thrombosis, or chronic endarteritis, one of the branches of the gastric artery is occluded. The corresponding portion of the wall of the stomach dies, and is destroyed by the action of the gastric juice.

Symptoms.—Pain is often present, continuons but with exacerbations, increased by food in the stomach, by motion, or by pressure; referred to the epigastrium, to the left hypochondrium, to points lower down in the abdomen, or to the back.

Vomiting is very common, but varies as to its frequency.

Blood is vomited in one-fourth of the cases. It is vomited in the form of coffee-ground matter, or of small clots, or in large quantities. The patients may bleed to death in a short time.

According to the extent of the chronic gastritis, there are symptoms of gastric indigestion.

The Course of the Disease.—There may be no symptoms during life, and the ulcer, or its cicatrix, is found after death from some other disease.

There are cases which last for weeks, months, or years. The symptoms are marked, but more or less severe. Some of the patients recover, others die of starvation.

There are cases in which one or more large hemorrhages form the prominent, and sometimes the only, symptom.

There are cases in which the large or small perforation of the wall of the stomach forms the prominent feature.

There are cases in which the deformity of the stomach, produced by the cicatrization of the ulcer, gives symptoms for the rest of the patient's life.

Treatment.—The first point is to determine whether the patients can be fed by the stomach or by the rectum. If they can be fed by the stomach, milk, peptonized milk, cream, beef juice, scraped beef, and koumyss are usually the best articles. If they have to be fed by the rectum, peptonized milk, cream, the yolks of eggs, blood, and beef juice with a little brandy and opium added, answer very well.

For drugs we employ the alkalies, bismuth, cocaine, oxalate of cerium, nitrate of silver, iodoform, hydrocyanic acid, or opium.

For external applications we employ heat, cold, the faradic current, and counter-irritation.

CANCER OF THE STOMACH.

Lesions.—The new growth, beginning in the glandular coat, takes the form of flattened tumors which ulcerate at their centres, or of polypoid tumors projecting into the cavity of the stomach, or of flat, diffuse infiltrations of the wall of the stomach. With any of these

forms there is regularly also more or less chronic gastritis.

Most of the tumors are situated at the pyloric end of the stomach, and may extend from there to the lesser or greater curvature, the anterior or the posterior wall. A smaller number of tumors are found at the œsophageal end of the stomach, and may extend up and involve the œsophagus. The pyloric tumors may occlude the pylorus and produce dilatation of the stomach. The peritoneal coat of the stomach may be inflamed and form adhesions with the surrounding viscera.

Secondary tumors are most common in the liver, the neighboring lymphatic glands, and the peritoneum; but they may be found in any part of the body.

Causes.—The disease is about equally common in males and females. The maximum liability is between the ages of sixty and seventy, but the disease is common enough between the ages of forty and fifty, and even between the ages of twenty and thirty it is occasionally met with.

Symptoms.—The appetite is poor. Nausea and vomiting are often present. The vomiting is of food, of brownish or yellow fluid, of coffee-ground matter, or of blood.

In some of the cases the vomiting is an early symptom, and continues throughout the disease. In some cases there is no vomiting till late in the disease. Or the vomiting may accompany the early stages of the disease and afterward stop. Or there may be no vomiting at all.

Pain referred to the stomach is a frequent, but by no means a constant, symptom.

A tumor is the most characteristic symptom of the disease. The ease with which it can be felt depends upon its position, its size, and its mobility. It is not until the tumor can be felt that the diagnosis is certain.

It is not uncommon for a patient to have chronic gastritis for some years before the development of the cancer. When this is the case the symptoms of the two diseases are of the same kind and are continuous.

In the later stages of the disease there may be a moderate rise of temperature.

The bowels are usually constipated, but there may be diarrhoea.

Not infrequently the patients are jaundiced. Sooner or later the patients lose flesh and strength, become anæmic and cachectic, and may have some œdema of the legs.

The ordinary duration of the disease is about one year, but some cases go on for two, or even three, years.

The *Diagnosis* is often difficult. The symptoms are much the same as those of chronic gastritis and of ulcer of the stomach. The presence of a tumor is the only positive diagnostic sign.

The *Treatment* consists in the regulation of the patient's diet, and the use of opium to alleviate pain. The removal of the tumor by operation, although it has been done many times, is not yet fairly established as a therapeutic measure.

THE INTESTINES.

THE small intestine is sometimes the seat of a catarrhal inflammation which may be of mild or of severe type.

Such an inflammation is especially common in young adults, some of whom seem to have a predisposition for the disease. In some cases there is a history of indigestible food, or of exposure to the weather ; in other cases no exciting cause can be discovered.

Symptoms.—The patients have marked and constant pain and tenderness referred to the lower part of the abdomen. There is a rapid rise of temperature from 100° to 104° F. There is marked prostration from the first, the patients being at once confined to bed. There may be vomiting, the bowels are constipated. The invasion of the disease is sudden, the patients often seem seriously ill, but yet they regularly recover. The fever subsides first, then the pain, and in one or two weeks the patients are well. Sometimes, however, the pain persists for a longer time after the subsidence of the other symptoms.

Diagnosis.—The disease may be mistaken for inflammation of the vermiform appendix, or for peritonitis.

Treatment.—The patients are to be kept in bed ; at first on a fluid diet ; when the fever has subsided, on meat principally. Continuous heat or cold should be

applied over the abdomen. At first, morphine and calomel together are given in small doses, later belladonna and ipecac. The bowels are to be moved every day by enemata.

CHOLERA MORBUS.

This name is given to an acute catarrhal inflammation of the gastro-intestinal tract, probably associated with the growth of pathogenic bacteria.

Lesions.—After death the mucous membrane of the stomach and intestines is found coated with mucus, congested, sometimes with little abscesses in the glandular coat.

Causes.—The disease is especially common in the hot weather of August and September. As exciting causes improper food and contaminated water are probable.

Symptoms.—The attacks are apt to come on in the night. There is first a feeling of abdominal oppression and of prostration. Then the patients empty the stomach of food and the intestines of fæces. After this there is frequent vomiting and purging of white or brownish fluid. There may be colicky abdominal pains, and painful contractions of the muscles of the abdomen and of the legs. The patients are anxious, restless, much prostrated, tormented by thirst, the skin cold, the body soon emaciated, the heart's action rapid and feeble. The patients often seem seriously ill, but yet as a rule recover. Occasionally, however, the disease proves fatal, especially in old or feeble persons.

Treatment.—The patients are to be kept in bed, hot fomentations are to be applied over the abdomen, and opium and stimulants are to be given according to the

indications of each case. The food should be in small quantities—koumyss, beef-juice, milk, or cream and water.

CHOLERA INFANTUM.

This name is given to a disease of young children which resembles cholera morbus, and is probably due to the growth in the intestines of pathogenic bacteria.

Lesions.—After death the intestines contain white or brownish fluid. The mucous membrane is pale, the solitary and agminated glands are sometimes swollen, sometimes ulcerated.

Causes.—The disease belongs to children under two years of age. It is most apt to attack those who, from bad food, bad air, or heat, have their intestinal tract already in an unhealthy condition.

Symptoms.—The disease may follow an ordinary diarrhoea, or begins suddenly with purging, vomiting, and prostration. Vomiting is usually, but not always, present, and varies as to its frequency and persistence. Purging is constant and frequent. The movements are at first fecal, afterward of white or brownish fluid. The patients rapidly lose flesh and strength, the heart's action is rapid and feeble, there is constant thirst, the skin is cold, the urine is diminished. The patients are always restless and miserable, some of them have muscular twitchings, general convulsions, alternating delirium and stupor.

The disease may not last for more than twenty-four hours, or it may continue for several weeks. It is always serious, and often fatal.

Treatment.—The children are to be fed with small quantities of koumyss, cream and water, wine whey,

beef-juice, or milk and barley-water. In the prolonged cases oil may be rubbed into the skin. The children should be kept as cool as possible, and sent to a different climate from that in which they have been taken sick. Of drugs the most reliable seem to be combinations of mercury, alkalies, and opium in small doses. Stimulants may be necessary.

CONSTIPATION.

In the healthy adult there should be a movement from the bowels once in each twenty-four hours. The fæces should be formed, of natural consistence and color. In some individuals the regular interval is shorter, in some it is rather longer, the character of the fæces remaining normal. If the bowels cease to move regularly, and the fecal matters accumulate in the colon, the patient becomes liable to a variety of disorders.

In the treatment of constipation it is important to determine its cause and then to manage it, principally by attention to the diet and mode of life.

In some persons constipation is merely due to the habit of only going to the water-closet when they feel the impulse to defecate, whether it is once a day or once a week. In many of these patients the temporary use of a simple laxative or of a glycerine enema, with the enforcement of a regular daily hour for defecation, is all that is necessary.

In some patients the constipation is due to chronic gastritis. If the gastritis is improved by treatment the constipation will disappear.

The constipation may be due to an insufficient production of bile. Then we must at first use the drugs

which increase the formation of bile—*ipécac*, *podophyllin*, *bichloride of mercury*, *sulphate of magnesia*, the *alkalies*, or the *mineral acids*. Later the patient must take sufficient exercise and gradually give up the use of the drugs.

The constipation may be due to general bad health or *anæmia*, and the treatment has to be directed principally to the relief of these conditions.

Improper food and drink may be the cause of the constipation. The patients require fruits, vegetables, and starches in considerable quantities. They must take sufficient water, or one of the alkaline waters, coffee, or beer.

The most difficult cases to manage are those in which the sensibility of the mucous coat of the colon is diminished and its muscular coat relaxed. In these patients we apply massage and electricity to the abdomen, enforce proper exercise and diet, and give *strychnia*, *aloes*, and *belladonna*.

RETENTION AND IMPACTION OF FÆCES.

1. In old persons there may be a large accumulation of hard, fecal matter in the rectum. The patients complain of constipation or *diarrhœa*. They lose strength and flesh ; if not relieved, they die, apparently from fecal poisoning. In order to empty the rectum it is necessary first to break up and dislodge the fecal mass with the finger or some instrument, then to use repeated *enemata*, and then *colocynth*, *aloes*, and *strychnia*.

2. In adults there may be an accumulation of fecal matter in the colon at some point above the rectum. This may only be attended with constipation, colicky

pains, flatulence, and a tumor; or there may be also localized pain and tenderness, fever and prostration.

By the use of calomel, castor-oil, and large enemata these fecal masses can be removed.

3. There are rare and obscure cases in which the patients have diarrhœa, lose flesh and strength, and die. After death no lesion is found, except the distention of the entire colon with fecal matter.

DIARRHŒA.

A person is said to have diarrhœa if he has every day several loose, fecal evacuations without tenesmus.

Causes.—Extremes of heat and cold, decided emotions, such as fear, and a variety of nervous influences can produce a temporary diarrhœa.

Some of the fruits and vegetables, especially if they are unripe or partly decayed, or any undigested portion of food, act as irritants to the intestine and produce diarrhœa.

Any inflammation or new growth of the colon is liable to produce diarrhœa.

Children are especially liable to diarrhœa.

The milder forms of diarrhœa in adults require but little treatment. Rest, a restricted diet, a laxative, a little opium are sufficient.

There is a peculiar form of diarrhœa in adults, which behaves as if it were caused by a subacute catarrhal colitis, which often lasts for years, and which is difficult to manage. The patients have nearly every morning several large fluid passages, and then during the day no further trouble. Or, similar diarrhœal passages may be repeated after each meal; or, in addition, the patients

have a good deal of abdominal pain. In the mild cases the general health hardly suffers; in the severe cases there is decided loss of flesh and strength. The disease continues for years, and comes back repeatedly after ceasing for a time.

An exclusive diet of milk, or of meat and hot water; change of climate; cold water enemata; castor-oil, arsenic, iron, quinine, salicylic acid, are among our most efficient methods of treatment.

INFANTILE DIARRHŒA.

Diarrhœa is a common disorder of infants and of young children. It is especially common during the period of dentition, in hot weather, and in bottle-fed children.

The diarrhœa may follow an acute gastritis, an acute colitis, or begin of itself. The children have every day a number of loose, bad-smelling, green, or light-colored fecal passages. There may be a febrile movement, especially in the afternoon. The patients gradually lose flesh and strength. Such a diarrhœa may last for days, weeks, or months.

Treatment.—If the children have a good nurse they continue to nurse. If not, they are fed on cream and water, milk diluted with a thin gruel, or koumyss. Older children can take beef-juice and scraped meat. For medicines opium, castor-oil, calomel, sulphate of magnesia, and rhubarb are the most efficient. If the disease continues care must be taken that the child gets enough food. Small doses of opium, castor-oil, ipecac, podophyllin, bismuth, and bichloride of mercury are of service.

Occasionally we see cases in which there is first an

ordinary diarrhœa. Then the passages become white, larger and larger, until the child passes three or four times a day enormous quantities of white, pasty fæces. The children are tormented by hunger and thirst, and become very much emaciated. They should be removed to as different a climate as possible from the one where they were taken sick. The most efficient medicine seems to be a combination of hydrochloric acid, arsenic, and opium.

INTESTINAL COLIC.

This name is given to attacks of pain due to spasmodic contraction of the muscular coat of the colon.

Such attacks may be due to the presence in the intestine of irritating pieces of food, or fecal matter. Or the attacks may come on without discoverable cause.

Symptoms.—The pain is apt to come on suddenly, its severity varies, it may be referred to any part of the abdomen. The abdominal wall is retracted and hard, or distended. There may be nausea and vomiting, the bowels are constipated until the end of the attack. During the attack the skin is cold, or bathed in perspiration; the temperature remains normal, or is raised.

The attacks vary as to their severity and duration. Some of them are very alarming.

Treatment.—The object of treatment is to relax the muscular spasm and to remove irritating substances from the intestine. To relax the muscular spasm we use hypodermic injections of morphine and belladonna, hot fomentations over the abdomen, and hot water enemata. To remove the irritating substances we give castor-oil.

CHRONIC INTESTINAL COLIC.

The patients have attacks of colic at first at long intervals, then at shorter intervals, finally nearly every day. With the pain there is more or less tenderness over the colon. The patients after a time lose flesh and strength, some of them acquire the opium habit. The disease continues for years, steadily getting worse ; between the attacks constipation and flatulence are often present.

Treatment.—The worst cases require for a time an absolute diet of meat and hot water. In the less severe cases it is sufficient to exclude milk, tea and coffee, beer, soup, and most of the fruits and vegetables. The most efficient drugs are ipecac and belladonna, given together at regular intervals. Opium must be discarded altogether.

COLITIS.

The colon may be the seat of acute or chronic catarrhal, croupous, or follicular inflammation. If the inflammation involves the rectum, and blood and mucus are passed, the patients are said to have dysentery. Or if the disease has the characters of an infectious inflammation, they are also said to have dysentery.

Causes.—Sporadic cases of colitis occur in all climates, they are especially common in August, September, and October. Contaminated water, food, and atmospheric conditions seem to act as exciting causes.

In tropical countries the disease is endemic. It may occur as a local epidemic anywhere. In armies it is developed frequently. It often behaves as if it were an infectious inflammation due to pathogenic bacteria.

ACUTE CATARRHAL COLITIS.

Lesions.—The mucous membrane is swollen, congested, it may bleed, the mucous glands produce an increased quantity of mucus. In severe cases there is an emigration of white blood-cells which infiltrate the mucous membrane and are mixed with the mucus. The inflammation may involve the rectum alone, the rectum and the lower part of the colon, or the upper part of the colon alone.

Symptoms.—If the rectum is inflamed there may or may not be first an ordinary diarrhoea. The inflammation of the rectum is accompanied by pain, an inclination to defecate, and the passage of small quantities of blood and mucus. The patients feel moderately sick, there is sometimes a rise of temperature. The inflammation regularly runs its course within a week, and is rarely fatal.

Treatment.—The patients are to be kept in bed and fed with milk, gruel, beef-juice, and scraped meat. The bowels are to be opened every other day by castor-oil or sulphate of magnesia; the pain is to be relieved by opium.

If the upper part of the colon alone is inflamed, the patients do not have the small, painful passages of mucus and blood. The passages are large, fluid, and partly fecal, sometimes with blood. There are colicky pains in the abdomen, prostration, and a febrile movement. In adults the disease usually lasts as long as two weeks, in children even longer. In the longer cases there is considerable loss of flesh and strength.

Treatment.—The patients are to be kept in bed, and

fed with beef-juice, scraped meat, and gruel ; milk is to be avoided. The bowels are, at first, to be moved with castor-oil or sulphate of magnesia. Later we give small doses of castor-oil, sulphate of magnesia, ipecac, belladonna, or opium.

ACUTE CROUPOUS COLITIS.

Lesions.—The mucous membrane is congested, swollen, infiltrated with fibrine and pus, and a layer of fibrine and pus is formed on its free surface. There is superficial or deep necrosis of the inflamed portions of the colon. The inflammation is sometimes diffuse, more frequently confined to a number of small, circumscribed portions of the mucous membrane. The inflammation may involve the rectum alone, the rectum with more or less of the colon, or the upper and middle portions of the colon without the rectum. After the inflammation has subsided superficial or deep ulcers are left, according to the extent of the necrosis.

This form of colitis is always severe and often fatal. It behaves as do the infectious inflammations.

Symptoms.—The patients have numerous small, painful passages of blood and mucus. If the inflammation extends above the rectum they also pass large quantities of brownish fluid. There is nearly constant pain, irritability, and a desire to go to stool. There may also be colicky pains, strangury, vomiting, and jaundice. There is regularly a febrile movement, sometimes preceded by chills, throughout the disease ; but in some of the bad cases the temperature is low throughout. In the severe cases the prostration is marked, the heart's action is rapid and feeble, delirium, stupor, or convulsions are

developed. The disease lasts for from one to four weeks.

If the inflammation involves the upper portions of the colon alone, the patients at first have a moderate diarrhoea. This diarrhoea continues, the patients lose appetite, flesh, and strength, they have fever in the afternoon. After one or two weeks they have one or more hemorrhages from the bowels. After this they have higher temperatures and get worse much more rapidly. The disease lasts for several weeks, and is very fatal.

Prognosis.—Croupous colitis is a very serious disease. It is often fatal; it is often followed by chronic colitis. It is especially fatal in old persons, in young children, in tropical countries, with epidemics, with inflammation of the entire length of the colon.

Treatment.—The patients are to be kept in bed and on a fluid diet; they may require stimulants. Of medicines, the most efficient are castor-oil, sulphate of magnesia, ipecac, and opium. The castor-oil and sulphate of magnesia are given at long intervals and in full doses, to empty the colon of fæces. They are given in small doses, at short intervals, to lessen the colitis. The ipecac is given once or twice a day, in doses of from ten to twenty grains. The opium is given to relieve pain.

FOLLICULAR COLITIS.

Lesions.—The lymphatic glands in the wall of the colon are inflamed, swollen, softened. They slough and leave small, round ulcers. Associated with these changes there will be more or less catarrhal or croupous inflammation of the mucous membrane between the ulcers.

The inflammation is apt to involve a large number of glands throughout the whole length of the colon.

Causes.—This form of colitis is especially common in armies.

Symptoms.—The patients have small, painful passages of blood and mucus, and large diarrhoeal passages. At first there is fever, the passages continue; the patients lose flesh and strength. The great danger of this form of colitis is its persistence.

Treatment.—Change of climate, the regulation of the diet, and the use of every means to improve the general health, are indicated.

CHRONIC CATARRHAL COLITIS.

Lesions.—The mucous membrane alone, or all the coats of the colon, are thickened. There may be little polypoid growths on the surface of the mucous membrane. There is an increased production of mucus. Such an inflammation may involve the rectum alone, or the colon also.

Symptoms.—The patients pass mucus in small or large quantities, blood, and loose fecal matter. There is often constipation. The patients steadily lose flesh and strength. The natural tendency of the disease is to continue, but with periods of improvement.

Treatment.—The patients do best in a dry inland climate. The food must be restricted, even an exclusive milk diet may be necessary. The best medicines are castor-oil in small doses, the mineral acids, and ipecac.

CHRONIC COLITIS WITH ULCERS.

Lesions.—The ulcers are small or large, numerous or few, close together or scattered, confined to the rectum or extending up on the colon. The mucous membrane between the ulcers is more or less changed by catarrhal inflammation. The walls and floor of the ulcers are formed of granulation tissue.

Causes.—The ulcers are regularly formed during the course of an acute croupous or follicular colitis.

Symptoms.—The patients have frequent passages of blood, of mucus, or of fecal matter. They steadily lose flesh and strength.

Treatment.—The patients are to be managed in the same way as those who suffer from chronic catarrhal colitis. But if the ulcers are situated low down in the rectum, there is also an advantage in the use of local applications to the ulcers. These applications may be made by suppositories, or by enemata, or the patient is etherized and the ulcers brought into view with the speculum.

MEMBRANOUS ENTERITIS.

This is a chronic catarrhal colitis attended with a large production of very tenacious mucus, which comes away with the stools in the form of masses, cords, or tubes looking like membranes.

Causes.—The disease belongs to middle life, and is more common in women than in men.

Symptoms.—So far as the colon is concerned, the symptoms are : constipation, sometimes diarrhoea, flatulence with its attendant pains, and the passage at varying intervals of the so-called membranes already mentioned.

But with these symptoms are usually associated complicating conditions which are often more distressing than the intestinal symptoms. There may be gastric dyspepsia; functional disorders of the liver, diseases of the uterus and ovaries, hysteria, or hypochondriasis.

Prognosis.—The disease is not a fatal one, but yet a considerable time may elapse before the patient entirely recovers.

Treatment.—In the more severe cases the patients must be put on milk diet, or the use of scraped beef and hot water. An out-of-door life in a dry inland climate is regularly of service.

The most useful drugs are the alkalies, the mineral acids, small doses of castor-oil, ipecac, and sometimes small doses of opium. There is also sometimes an advantage in the use of large enemata of cold water, and of massage of the abdomen.

CARCINOMA OF THE COLON.

The new-growth usually follows the type of colloid cancer, or of glandular cancer with cylindrical cells. The favorite situations are the rectum and the caput coli. The new-growth surrounds the gut and extends longitudinally for one or two inches; less frequently it involves a considerable part of the length of the colon. The growth may cause a stricture of the colon, or may project inward in soft masses, or may ulcerate. It often infiltrates the surrounding tissues, it may cause a local peritonitis, or the ulceration may go on to perforate the wall of the colon.

Causes.—The disease occurs regularly in persons over thirty years of age.

Symptoms.—If the growth is situated in the rectum the first symptoms are constipation, flatulence, pain, and constricted stools ; or diarrhœa and passages of blood and mucus. As the disease continues the local symptoms become more marked, and the patients lose flesh and strength and become cachectic. The tumor can be felt in the rectum as a hard ring constricting the gut, or as a hard diffuse mass about the gut, or as a soft tumor projecting inward.

As the disease goes on the stricture becomes tighter and tighter ; or there is paralysis of the sphincter with a constant flow of brown, sanious fluid.

The patients die worn out with the disease, or with retention of fæces, or from perforation of the intestine, or from peritonitis.

If the growth is situated in the caput coli the first symptoms are gradual loss of flesh and strength, flatulence, alternating diarrhœa and constipation, nausea, and vomiting. Or the first symptom is pain coming on in attacks, first at long intervals, then more and more frequently. These symptoms continue, the patients lose flesh and strength, and become cachectic ; there may be distention of the small intestine. The tumor can be felt in the right side of the abdomen, when it has reached a sufficient size.

Occasionally we see cases of cancer of the rectum, or of the caput coli, in which there are no local symptoms. The only evidences of the disease are the loss of flesh and strength, and the tumor.

Treatment.—The only efficient treatment is the removal of the tumor by a surgical operation.

